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POSTURAL ACTIVITY OF MUSCLE AND NERVE.

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I.—INTRODUCTORY.

PROMINENT in the volume of *Brain* for 1888 is a discussion on Muscular Hypertonicity. At the opening of that discussion Hughes Bennett furnished a definition of "reflex muscular tonicity" as "that slight constant tension which is characteristic of healthy muscle." Thus simply expressed that is, I think, the current conception which still generally prevails. Simple though it is, it is one which a teacher of physiology finds difficulty in illustrating to students, and the statement remains vague to them. The almost complete absence of numerical measures of tonus, the lack of assignment to it of any clearly purposeful rôle, the want of demonstration of it in the various laboratory exercises on muscle—almost always excised frog's muscle—imbue the phenomenon with a certain mystery unwelcome to those desirous of clear facts for direct use in future practice. To tell the inquirer that tonus serves to obviate the muscle's "taking up slack" when it enters on the performance of a movement, seems rather thin-spun when the class-room exercise has measured the latency of toneless frog muscle as one-hundredth of a second. The further suggestion that the katabolism accompanying the tonus makes it an important means of heat-production—the so-called chemical or thermal tonus—suggests that the contraction tonus is mere by-play in the thermotaxis of the organism; and the explanation comes all the more as a surprise since the chemical

turn-over adjunct to the mechanical tonus has been shown in recent years to be very small.

Renewed inquiry on a larger basis is now lending more precision to the phenomenon of tonus. But, as is natural in a phase of transition, some confusion obtains. That this is so a quotation may show. Writing recently, Matula [21] says: "By reflex tonus is understood in this paper that persistent weak activity of the musculature which is excited by the stimuli continually reaching the body, that is, 'tonus' in Brondgeest's old sense. I find it necessary to state this although perhaps obvious, for the word 'tonus' is certainly one of the most misused terms in physiology. Under it each writer understands something different, if indeed he is in a position to supply any sharp definition at all of the conception."

The confusion seems partly traceable to want of touch between inquirers engaged upon the phenomenon in fields of observation seemingly rather wide apart. The publications of zoologists and clinicians do not commonly meet quickly. An aim of the present article is to try to correlate a number of such observations and tentatively to piece them together.

A fairly literal meaning attaching to the term "tonus" is, of course, "mechanical tension." In this sense it fits well the slight steady enduring tension so characteristic of muscles in their state of reflex tonicity. This meaning of the term is evident in the definition quoted from Hughes Bennett. But in its early use, by J. Müller and writers of that period, the term carried also or soon came to carry the implication of "automatism."

One meets the term in Humboldt's [15] "Researches on Muscle and Nerve" of 1797; there it is applied to nerve, and though used only occasionally, evidently refers to some then current view which postulates an intrinsic activity of the nerves and nervous system generally. That meaning outlived considerably the discovery that the tonus of which Müller [27] spoke was reflex and not the outcome of automatism of nerve-centres. The connotation of the term has drifted in various directions from the literal meaning of mechanical tension. It is applied to the activity of nerve-centres, thus: "tonic nerve-centres," "tonic discharge of nerve-cells" and the "bio-tonus" of living matter. In these departures there is always more or less prevalent the signification "lasting" and "enduring" muscular or nervous action as contrasted with evanescent or passing. To-day we commonly speak of the action of a muscle or nerve as tonic when we wish to indicate that it is rela-

tively long-lasting. The term has become somewhat vague by reason of the multiplicity of meanings attached to it. But one service expected of a technical term is that it should be precise and unequivocal.

And the conception itself, as well as the term, labours in one important respect under vagueness. Physiology pursues analysis of the reactions of the body considered as physical and chemical events: but, further, it aims at giving reasoned accounts of the acts of an organism in respect of their purpose and use to the organism *qua* organism. This may be called a teleological aim, yet belongs to a teleology not foreign to the scope of natural science. In animal behaviour the more complex the act the less equivocal usually its biological meaning. The physiologist in analysing animal reactions seeks, as a rule, components more elementary than those toward which the "behaviourist" directs his work. Thus, the "behaviourist" examines a train of acts characterizing some instinct; he seeks to describe the sequence of events from their outset in initiatory stimuli onward to movements and turning-points of movements often each one of high complexity and intricate co-ordination, and yet each one sufficiently unitary to serve for his present purpose as one separable piece of the train of behaviour he is fractionating. The physiologist aims at yet simpler units, for instance at the characteristics of a synaptic function, or the dissociation of the periodicity of a nerve-centre's activity from that of a muscle's activity. He takes for his problem reflexes usually much simpler than those studied by the "behaviourist." He may choose some reaction which itself is but a partial factor in a single act a whole train of which form the subject-matter of the "behaviourist." Yet every reflex is in its own measure an integral reaction, and is purposive in that it bears some biological purport for its organism. Every reflex can, therefore, be regarded from the point of view of what may be called its "aim." To glimpse at the aim of a reflex is to gain hints for further experimentation on it. Such a clue to purpose is often difficult to get; and attribution of a wrong meaning may be worse than absence of all clue. But the difficulty is generally inversely as the complexity of the reflex. Thus the larger the muscular field involved in the reflex effect the plainer usually its purpose. A slight movement confined to a single limb or, in appearance, to a single muscle, a transient rise of arterial pressure; these observed alone lie open to many interpretations and admit of no security of inference. They are fractional reactions which may belong to any of many general reactions of varied aim. Thus reflexes observed in paraplegic man have been notoriously difficult

to refer to their functional purpose. On the other hand, in lower animals where depression of spinal function and spinal shock are less, the ampler reflex actions, embracing by irradiation wider groups of associated muscles, often write their own meaning clear, and can indeed give a clue to the meaning of the analogous reactions less obviously decipherable in man.

When the spinal dog in response to an irritative stimulus of the scapular skin brings the hind foot to the irritated point and scratches there, the purpose of the reaction is clear. And a reflex of simple kind may have as much purposive completeness as a complex one. Thus, the reflex emptying a viscus, though it involves but a restricted field of musculature, may be as complete for its purpose as is such a reflex as walking, which involves skeletal musculature practically throughout the body.

Further, among data helpful for assigning its purpose to a reflex is the fashion of its elicitation, the nature of the adequate stimulus. That a faradic stimulus applied to the back of the tongue in a decerebrate cat interrupts the rhythm of the respiratory movements, is an observation which may leave us in doubt as to the biological meaning of the result. But the same result, when caused by putting a few drops of water on the back of the tongue, suggests an obvious "purpose," and the interpretation is clinched by the reflex swallow which ensues.

As to muscular tonus much of it is reflex. To know the biological purpose of such a reflex reaction is to have suggestions for lines along which to investigate it. The question arises, does muscular tonus carry the same biological meaning in all its examples, or does it in some cases meet one purpose, in other cases meet another? The decipherment of what biological meanings its various instances possess should help toward obtaining a broader standpoint for evaluating the whole phenomenon itself. A step toward this is to consider manifestations of it in particular cases. In the first place a field of musculature may be taken which is skeletal, because although complex its very specialization reveals the more clearly the particular purpose it effects.

II.—Skeletal Muscle.

By Johannes Müller the term "tonus" was employed to denote a certain steady slight contractile tension which he regarded as the characteristic condition of normal skeletal muscle when not engaged

in the performance of any specific act. He thought this slight steady tension the outcome of an influence continually imparted from the nerve-centres of cord or brain. Whether this action of the nerve-centres was of automatic or reflex production he left open. Marshall Hall [14] considered the spinal influence maintaining the steady closure of openings by sphincters to be a reflex tonus, though, it is true, he gave no further account of its source or of the stimulus exciting it.

The early experiments directed toward demonstrating the state of tonus of skeletal muscle met with divergent results. Their plan of observation was to look for a slight elongation of the muscle (frog) after severance of the muscle's nerve. Heidenhain, Auerbach, Schwalbe and Pflüger were among those who failed to get clear evidence of any such lengthening. In the fifties of the last century the existence of "tonus" of skeletal muscle seems to have become doubted by many physiologists. But in 1860 it was reaffirmed by the observations of Brondgeest [7], of Utrecht. Brondgeest showed that in the frog if the afferent spinal roots of one hind limb be severed, the corresponding limb when the frog is held vertical is less flexed than is the fellow sound limb. He argued that the greater flexion of the sound limb is due to a reflex tonus of its muscles. The observation was soon recognized as trustworthy. The failure of previous observers to find any definite elongation of the skeletal muscle on nerve severance remained, however, unaccounted for. Twenty years later a critical summary of the position by Eckhard [11] concluded that "tonus" of skeletal muscle had been demonstrated for but few muscles, and was far from having been shown present in all muscles and at all times. As to the source of reflex tonus in those muscles in which it was present Eckhard inclined to accept the suggestion that the source is cutaneous, the skin at one point or another being continually open to numbers of stimuli, which, although not individually sufficient to cause reflex movement, yet by their summation keep up a more or less continuous additive mild reflex effect. And the skin as a source of the reflex tonus of skeletal muscle is still apparently occasionally accepted.[1] But Mommsen [24], in 1885, pointed out that the Brondgeest phenomenon persists after the limb has been stripped of its skin.

The tonus of skeletal muscle can be studied favourably in the mammalian preparation. Removal of the brain from the posterior colliculi forwards in the cat provides an excellent tonic preparation of

[1] W. Fröhlich. *Zeitschr. f. Allg. Biologie*, 1911, ix, 1, 63.

the extensor muscle of the knee. This tonus is still retained by the muscle to the full after severance of all the skin nerves of both hind limbs. Further, the muscle still retains its full tonus after severance of *all* nerves of both limbs excepting only the nerve of the tonic muscle itself. That nerve consists, of course, of fibres afferent as well as efferent. The afferent fibres are traceable partly from the muscle's tendon, but mainly from the muscle itself. These afferent fibres reach the spinal cord *via* the dorsal (posterior) roots of two spinal nerves (the fifth and sixth lumbar in the cat). If these two afferent dorsal roots are severed, the tonus at once vanishes from the muscle, although the corresponding ventral roots containing the motor fibres for the muscle remain intact, and although all the other nerves of the limbs remain intact as well. And similar experiments with other muscles exhibiting tonus, e.g., gastrocnemius, semimembranosus, triceps, supraspinatus, meet the same result. In each case the tonus of these muscles requires the afferent fibres of the tonic muscle itself, and in the decerebrate cat preparation no other afferent fibres than those of the tonic muscle itself are actually essential for the exhibition of the tonus.

The tonus of these muscles in this decerebrate preparation is not a phenomenon requiring for its detection and demonstration any refined apparatus, or indeed any apparatus at all. It and its features are palpable and obvious; graphic records of it are obtainable by relatively coarse methods. The extensor muscle of the knee, vasto-crureus, lends itself well to the purpose. That and the three other muscles mentioned above are specimens of the tonic muscles in this preparation. But many other muscles in this preparation show the tonicity as well In the hind limb the distribution of the tonicity in the musculature shows the following feature. If the reflex act of stepping is examined as it may be both in the spinal and in the decerebrate preparation (cat), the act is found to consist of two phases; in one phase—the flexion phase—the foot is lifted slightly from the ground, and the limb is swept forward by flexion of hip, accompanied by flexion at knee and ankle, so that the foot may clear the ground in its advance. In this phase all the flexor muscles of the limb are excited to contract, and all its extensor muscles are inhibited by reflex inhibition. The other phase—the extension phase—is that in which the foot being in contact with the ground, the limb is straightened at knee and ankle, and kept from bending under the body's weight by the extensors of those joints; and at the hip, the extensors using the foot's *point d'appui* against the

ground as a fulcrum for the leverage by the limb, push the body forward. In this phase all the extensors of the limb are in active contraction, and the flexors are reflexly inhibited. The distribution of the reflex tonus of the decerebrate preparation in the musculature of the limb is exactly to those muscles which are in active contraction in the extensor phase of the step, to those and to no others.

In the forelimb, though this analysis of the exact distribution in the musculature of the extensor phase of the step has not been so completely made, the analysis, so far as it goes, shows again exact correspondence between the musculature exhibiting the tonus and those engaged in contracting in the extensor phase of the step. The two phenomena involve, and are confined to the same group of muscles. It is in the extensor phase of the step that the limb is supporting the weight of the body.

The distribution of the tonus in the limb musculature reveals, therefore, arrangement on a plan of strict co-ordination. It is, however, not confined to the musculature of the limbs. It is as markedly present in various other regions. In the trunk it obtains in the muscles which bend the vertebral column upwards (opisthotonos); in the neck in those muscles which lift (retract) the neck and head; in the caudal region in those which lift the tail; in the head in those which close the jaw. It is not present in those muscles which bend the spine downwards, droop the neck and tail, flex the head, depress the jaw. It seems to be present, but of this I am not entirely sure, in the ventral muscles of the abdominal wall. Evidently, therefore, the distribution of this reflex tonicity embraces just those muscles whose contraction tends in the erect position of the animal to counteract the effect of gravity on the various several regions, the muscles which prevent those parts, and the animal as a whole, from sinking to the ground. And from the muscles antagonistic to these the reflex tonicity is absent. In other words, the reflex tonus obtains in, and is confined to, those muscles which maintain the animal in an erect attitude. That this is so may be demonstrated by setting the decerebrate preparation on its feet; it is then seen that the preparation stands. Thus this reflex tonicity, which when seen in a single isolated muscle prepared for the myograph, does not carry on the face of it any very obvious biological purpose, does carry a clear and unmistakeable biological purpose when the phenomenon is followed in the musculature as a whole. The reflex tonus is postural contraction. Decerebrate rigidity is simply reflex standing. The reflex tonicity of the skeletal

muscles of the decerebrate cat and dog is shown by its co-ordination, its effects, and its distribution in the musculature, to be a reflex which differs from the reflexes more commonly studied mainly in this, that the latter execute *movements* while this maintains *posture*. The reflex tonus is in short reflex posture, and in this case the posture maintained is that of *standing*. And the reflex posture is modifiable on the supervention of certain additional stimuli, and the modifications of posture thus obtained are so intelligible as forms of standing adapted to particular purposes that they carry on the face of them that significance. If the head of the reflexly standing decerebrate preparation be forcibly flexed, the postural contraction of the extensor muscles of the fore limbs is inhibited, and the animal's fore quarters sink, while at the same time the postural contraction of the extensors of the hind limbs increases, raising the hind quarters. The preparation thus assumes the attitude of a cat looking under a shelf. On the contrary, if the head of the preparation is passively tilted up and back the postural contraction of the extensor muscles of the fore limbs increases, raising the fore quarters, and at the same time the postural contraction of the extensors of the hind limbs is diminished so that the hind quarters sink. The preparation thus assumes the posture of a cat looking up to a shelf. There goes further with each main posture of the head even passively imposed upon the decerebrate preparation a corresponding reflex modification of the reflex posture of the limbs. Magnus and de Kleijn [20], to whom is owing the elucidation of this subject, have described these fully, and shown their constancy, and shown further that the centripetal impulses causing these reflex modifications of the reflex standing are traceable for their one part to the otic labyrinth, for their other part to the deep afferent nerves (proprioceptors) of the neck itself. These experimenters have succeeded in separating the reflex results from the two sources, and have thus determined what part each source plays in the combined effects which under natural conditions are those of usual occurrence.

As mentioned above, the afferent nerves producing and maintaining this postural reflex of standing are the afferent nerves of the posturally contracting muscles themselves. The whole reflex posture of standing is thus one great compound reflex built up of a number of component reflexes. To the making of the total reflex posture there goes the postural reflex of each limb, similarly the postural reflex of the neck, of the trunk, of the tail, of the head. And the reflex posture of each component region is in large measure separable from that of the rest,

and is capable, within limits, of modification, although remaining still contributory to the general posture of standing. This is in accord with the natural occurrence of, for instance, such a modification of the erect posture as "sitting" in the cat or rabbit; a posture half-way between lying down and standing, the fore limbs "standing" and the hind limbs lying down.

And the local reflex posture, say of a limb, can be modified by local influences. It is in the examination of these that we meet with exemplification of characteristics of postural contraction to which attention will be drawn in dealing with the postural contraction of muscular walls of the hollow viscera and blood-vessels. In the posturally acting skeletal muscles these characters appear as what have been termed [36] the "lengthening" and "shortening reactions." The "lengthening reaction" and the "shortening reaction" are given by the skeletal muscles of the cat in postural contraction and they are given also by the smooth muscle of the viscera, and of invertebrata. Just as the postural configuration of the knee or elbow is adjustable by these means so likewise is that of the bladder or the stomach.

The "lengthening reaction" of a skeletal muscle engaged in postural action is easily seen in the knee-extensor of the decerebrate preparation and can be suitably examined there. The muscle is isolated by paralysing, by appropriate nerve-section, all the other muscles of both hind limbs. As mentioned above, the muscle (vasto-crureus) when this has been done still maintains its own postural reflex action unabated. For the purposes of the observation the preparation is preferably placed on its side, on the left side if the vasto-crureus chosen be the right. The muscle still retains uninjured its attachment to the patella and, by patellar tendon, to the tibia. Starting with a knee-posture of nearly full extension the tension of the muscle as it holds the knee in this posture is examined by finding what weight pulling the tibia backward—i.e., tending to flex the knee—the postural action of the muscle just counteracts. The observer then with his hand forcibly bends the knee, overcoming the contraction of the extensor; he brings it into a new position, let us say, of nearly full flexion, performing the movement steadily and not too quickly—e.g., so that it occupies a couple of seconds. On then releasing the knee there is usually a slight brief partial movement of it in the direction of return toward extension, but apart from this, which does not alway occur, the limb remains in the new position, the position which was passively imposed upon it. The tensive pull of the extensor muscle as examined

by the weight, is in the new posture practically just what it was before, neither more tension nor less tension than before. But in the new position the length of the muscle is of course greater than it was in the former. In short, the posturally acting muscle has, in result of the extension imposed upon it, assumed a new postural length, and has assumed a greater postural length without exhibiting in its new state of greater elongation any practical departure from its previous degree of tension.

In the same preparation, conversely the shortening reaction may be elicited. For this, starting with nearly full flexion as the reflex posture of the knee, such a flexed posture for instance as is left at the end of a " lengthening reaction " like that just described, the tension of the muscle is estimated by the amount of load-traction it counterbalances on the tibia. The observer then passively extends the knee so as to bring it to, for instance, nearly full extension. On then releasing the limb it is found, although the weight is still exerting practically the same pull toward flexion as before, to remain in the new posture of extension passively imposed upon it. The posturally acting muscle has taken up a new postural length, and is now shorter than before, but exhibits practically the same tension as at its previous length. On examining the tension by the weight it just counteracts, there is found to be practically no alteration of the tension. The skeletal musculature by reason of these lengthening and shortening reactions allows that latitude of pose which is so useful and familiar a feature in natural attitudes. The animal may stand with right foot in advance of left, or left in advance of right, or with the two feet abreast of each other ; all these differences in detail from local posture to local posture are compatible with the general posture of standing in the animal as a whole. The postural reflex contraction is plastic in this sense, hence the term *plastic tonus* [36] has been applied to it. The skeletal muscle in this form of reflex contraction can quite readily adjust itself to different lengths while counteracting one and the same load.

In the above examples of the lengthening and shortening reactions of skeletal muscle, the alterations in posture, to which the limb was subjected in showing its power of adjustment, were alterations imposed by passive movement. The observer moved the limb into the new attitude which the reflex postural contraction then took up and lightly fixed. But the reflex adaptation to the new posture occurs just as well, or better, when the changed position of the joint is brought about by active reflex movement excited, for instance, by faradizing an afferent nerve-trunk. Thus in the postural reflex preparation when a reflex

extension movement of the knee has been provoked, the shortening reaction appends itself to the reflex contraction, and on discontinuing the stimulus which caused the extension movement the extensor muscle still remains shortened, and the knee still continues in the extended position. This lends to reflexes of the decerebrate preparation a character recalling some features of catalepsy. The limb brought by active or passive movement into a new posture when released remains in that new posture.

Again, the animal being on its side and the postural contraction of vasto-crureus just counteracting a certain weight tending to flex the knee by a cord running over a pulley and attached to the tibia, let an afferent nerve, appropriate for reflexly inhibiting the postural contraction of the muscle, be stimulated with a series of mild break shocks at equable intervals. Let the intensity of the stimulation chosen be such that each shock causes a minimal brief inhibitory lengthening of the posturally contracted muscle. The muscle elongates in a gradual minutely step-like manner. Throughout the steps of the lengthening process, and at the end of that process, when the electrical stimulation is withdrawn and a considerable total lengthening of the muscle has taken place, the postural contraction still just counteracts the same weight as it had just counteracted initially at the outset of the observation. The "lengthening reaction" obtains, therefore, with reflex inhibitory lengthening of the muscle as well as under forcible passive lengthening of the muscle.

That the *tension* of the posturally acting muscle does remain approximately unaltered over a large range of different lengths of the muscle in different postures of the joint is shown for the vasto-crureus by such experiments as the following. The preparation being on its side and the vasto-crureus the only muscle unparalysed by nerve-section, its postural contraction just counteracted a pull of 42 grm. on the tibia, 11 cm. from the knee-joint axis when the knee was at an angle of 110°, and when the knee was then flexed to 60° the postural contraction just counteracted a pull of 42·5 grm. at the same leverage.

The tension of the muscle has been examined by v. Uexküll and Noyons [28, 39] with sclerometers, instruments for determining the hardness of the muscle in its fleshy part. One pattern of sclerometer measures the amount to which a conical button when loaded with a weight indents the muscle—the *statical sclerometer*. This was applied to the fleshy part of vasto-crureus when the knee was flexed to different angles, the muscle being in postural contraction, but under the same

load in the different attitudes of knee. The hardness of the muscle as actually measured was practically the same in all the varied lengths of muscle under the different poses of the joint.

The observations on the tonus of skeletal muscle in the mammal, therefore, go to show that the phenomenon is in skeletal muscle nothing more nor less than postural contraction. Why did the search for tonus in the skeletal muscles of the frog meet with such discrepant results and equivocal interpretation? In that search it seems to have been regarded as unimportant what particular muscle was selected for the search, the supposition being implicit that tonus would attach either to all muscles or to none. But if we regard tonus as a contraction of muscles engaged in the execution of a definite co-ordinate reflex, a reflex differing from reflexes ordinarily examined only in its function being posture and not movement, we shall expect reciprocal innervation of antagonistic muscles to obtain, and in that case if one set of muscles are in reflex contraction their opponents will not be in contraction but under reflex inhibition. That in postural reflexes reciprocal innervation of antagonists does hold has been found in fact to be the case. Thus in reflex standing the opponents of the posturally contracting anti-gravity muscles, the flexors as they may in brief be termed, exhibit no postural contraction, and the stronger the reflex posture the less trace of contraction may these latter be expected to show. It is noteworthy that in Brondgeest's [7] experiment demonstrating reflex tonus in the hind limbs of the frog, the muscles which exhibit the tonus are *flexors*, the exact reverse of what obtains in the decerebrate cat, dog, and monkey. The habitual natural posture of the frog is squatting, not standing. In this squatting the head is somewhat lifted, the fore limbs are extended, propping the weight of the forepart of the animal, but the hind limbs are folded in nearly full flexion, doubtless in appropriate readiness for the hop. The spinal frog retains as its reflex attitude the posture of squatting. It was long ago noted by Volkmann that after destruction of the brain and upper part of the spinal cord in the frog, a sign that the immediate shock of the operation has subsided is that the hind limbs assume a folded position. In short, the flexed posture of the hind limbs natural to the ordinary squatting attitude is by the spinal frog retained as a spinal reflex. Since it is the flexor muscles which execute this posture, and since reciprocal innervation of antagonists obtains in reflex posture, search for postural contraction is foredoomed to failure if made in the extensor muscles of the frog's hind limb. But it was to the gastrocnemius, a main extensor muscle,

that Heidenhain, Jurgenssen, and others turned in their search in the frog for the tonus of skeletal muscle postulated by J. Müller. They failed to find it there. Their failure and Brondgeest's success in his experiment which dealt with the flexor muscles is therefore simply confirmatory of the view that tonus is postural contraction.

In the reflex standing of cat, dog, and monkey, it is the extensor muscles of the limbs which exhibit postural contraction; in the reflex squatting of the frog it is, as regards the hind limbs, the flexor muscles which exhibit it. Another instance of postural contraction exhibited by a flexor group of muscles is furnished by the wing of the pigeon as folded when the birds stands and walks.

In some invertebrates there seem to exist certain muscles concerned with posture solely. Thus in Echinus each "spine" is provided with a muscle which produces the movement of the spine and with another muscle which preserves when required the posture into which the spine has been brought by its movement muscle. The spine is mounted on a ball-and-socket joint, and on this by means of the outer capsular (movement) muscle it executes gyrating motion. If in the execution of this it meets with a resistance too great for it to overcome, the inner sheet of muscle is brought into play and fixes the spine in that position to which the movement muscle has brought it. The movement muscle is of long transparent fibres, the postural or check muscle (Professor Bayliss [1A] introduces the appropriate term 'catch'-muscle) of short opaque fibres. The postural fixation of the spines is of importance for the locomotion of the animal; as Echinus creeps forward under the pull of its tube-feet the postural fixation of the spines makes of the latter levers propping the weight of the body much as the limb extensors do in mammalian locomotion, save that in the latter case the motile agent is a push rather than a pull.

Another example of similar differentiation is furnished by the mollusca. In the bivalved molluscs an elastic ligament opens the shell, but muscles shut it. Of these latter there are combined for this purpose two kinds, the one of striped fibres, the other of unstriped. The former are those which produce the closing movement, the latter those which retain the posture of the shell at any degree of closure complete or incomplete to which the former muscle has moved it. The former are movement muscles, the latter postural muscles. By the former is produced the sudden adduction of the shell-valves which causes a quick current of water outward, sweeping with it anything harmful that may have entered the shell chamber. In Pecten, a form

specially studied by v. Uexküll [39] and J. Parnas [30], the movement muscle executes also the rhythmic adduction of the shell-valves in swimming. Pecten being a monomyarian has in appearance only a single adductor muscle, but examination shows this to consist of two parts, one the glassy "movement muscle" the other the semi-opaque "postural muscle." How completely the closed posture of the shell depends upon the latter is demonstrated by severing the visceral nerves supplying the postural muscle. The "movement" adductor muscle remains still unparalysed. The shell then stands open, and to various stimuli, which normally cause the shell to shut and remain shut, the shell responds more readily than usual by closure, but the closed posture is not maintained, and the shell gapes again at once.

With the intact Pecten, if a penholder, put between the open lips of the shell, touches the parts within, the shell-valves close upon the intruder and hold it tight. The wooden holder is so tightly gripped that it can be extracted only with difficulty, but that done the shell-valves remain at the same degree of partial closure, they do not close further. Yet any attempt to pull them farther apart reveals that to that they oppose a very great resistance. The movement of adduction was executed by the contraction of the "movement adductors": their short-lasting contraction then ceased, but the degree of adduction of the shell-valves thus arrived at is then maintained as a posture by the postural contraction of the postural muscle. The speed with which the postural muscle can by contraction shorten its length is relatively slow as compared with that of the movement muscle. But the postural muscle exhibits the phenomenon already adverted to in the extensor muscles of the limbs and neck in postural contraction, namely, the "shortening reaction," seen also in sipunculus and in the contractile skin of holothuria. If the shell-valves of Pecten be passively partly closed or fully closed they remain so. Their postural adductor takes up and retains the closed shell posture passively imposed upon it. It does so also when the shell is actively adducted by the movement adductor. V. Uexküll discovered a means of exciting a reflex opening of the shell. Experimenting upon this, he found that lengthening of the check-adductor in its postural action can be brought about reflexly.

The mechanical elasticity of the postural adductor is very small. Suppose a partially open Pecten after severance of the movement adductor and loaded with a weight so attached as to tend to pull it open farther, though failing to do so. On cutting the string attaching the weight, the shells do not perceptibly spring nearer together. Nor is

there any proportion between the power to move the weight and the power to support the weight. The postural adductor can support for hours a weight it cannot raise. A weakly Pecten which in the aquarium does not and cannot close its shell is, if the shells be closed by the observer and kept in the closed posture for a few minutes, found able then to keep the shells in that closed posture against the pull of the elastic opening ligament.

The existence in various invertebrata of muscles separately differentiated for execution of movements and for maintenance of posture respectively seems without parallel in the skeletal musculature of vertebrates. In the latter, one and the same muscle is used for the two purposes, though some muscles are predominantly concerned with the one, some with the other function. Perhaps the nearest approach to muscles of purely postural function in mammals are the sphincter muscles controlling orifices. But in most of the more complex reflex acts the reflex while employing some muscles for execution of movement simultaneously employ others for maintaining posture. Thus, in the scratch reflex while one hind limb is engaged in the rhythmic scratching movement, the reflex employs the muscles of the other three limbs and of the neck and head for the maintenance of a characteristic posture which continues so long as the reflex scratching continues. Similarly, in a powerful nociceptive flexion reflex, while the stimulated limb maintains the attitude of flexion the other limbs frequently perform stepping movements, just as in the intact animal that has stepped on a thorn the injured foot is held folded up and the other legs run away. And with these skeletal muscles one and the same muscle may at the same time exhibit both postural contraction and phasic or movement contractions. Thus, in the scratch reflex, there is required in order that the hind limb reach the neck and apply there its scratching movement a certain posturing of the limb as well as rhythmic movement of it. For this there is demanded some postural flexion of the hip. It is found that the sartorius muscle, which is a flexor of hip and knee, shows in this case a well-marked degree of steady postural contraction as well as, over and above that, the characteristic four per second rhythmic contraction of the scratching movement itself.

A question which arises is whether in all cases the reflex postural action of skeletal muscle depends normally upon the afferent nerve of the posturing muscle itself. In the reflex posture of standing exhibited by the decerebrate cat that does largely seem to be the case. Severance of the afferent spinal roots of both hind limbs in the dog (Bickel [2]),

renders standing with those limbs impossible for a considerable period; but in the course of time the animal becomes able to support itself upon them, a compensation traceable to labyrinths, cerebellum and motor region of cerebral cortex. In the pigeon, severance of the afferent roots of both hind limbs makes standing impossible (Trendelenburg). Severance of the afferent roots of one leg impairs also the flexion posture assumed by the hind limb during flight. Section of the afferent roots of the wing affects little, if at all, the folded posture maintained by the wing when not in flight—e.g., during standing or walking. Nor has the source of the postural contraction of the flexors of the wing been found. Here, as in the case of the iris, the postural contraction if, as is presumable, reflex, lies in receptors not those of the contracting muscles themselves, is, in short, allogenous not autogenous.

That receptors other than those of the contracting muscles themselves can be adjuvant to the reflex postural action maintained by these latter is evidenced in many observations. For instance, Ewald [12] has shown that the postural closure of the pigeon's beak is impaired by destruction of the labyrinth; and after splitting the lower bill into its two lateral halves he found that destruction of the right labyrinth weakened the postural contraction of the right half much more than that of the left: the movement of closure was often fully executed, but with the right half the closed posture was less powerfully maintained; this was clearly demonstrable by hanging upon each of the separate halves one of a pair of equal weights—the right half did not maintain the closed posture under so heavy a load as did the left: an observation recalling those above mentioned on the pecten shell.

And Magnus and de Kleijn [20] have shown the existence of a number of important postural reflexes of labyrinthine origin affecting the extensor muscles of the limbs. To excite the labyrinth they employed modes of stimulation natural to it—"adequate" stimuli—namely posings of the head in regard to the direction of gravitational force. The labyrinth is a receptive organ specialized for reacting to gravitation force, hence it initiates sensations reporting on the spatial relation of the head to axes running through it and the earth's centre, in short, to the vertical, and the reflexes which the labyrinth initiates consist in adjustments of the head, including eyeballs and jaw, to the vertical. The action of the muscles in the posture of standing is anti-gravitational. Hence it is not surprising that the labyrinth reactions should be related to and influence the anti-gravitational reflex of standing, though this latter has its origin in the receptive organs of the standing

muscles themselves. Magnus and de Kleijn show that the labyrinthine influence on the postural contraction of the extensors of the limbs is adjuvant and symmetrical for each limb-pair, that is, right and left fore limb are affected similarly and together, and right and left hind limb similarly and together. They show that the adjuvant influence is greatest when the head is inverted, least when the head is right side up, and that in lateral positions of the head the influence is of intermediate degree. In these experiments the influence of neck posture was excluded by fixating the neck in plaster of Paris or by severance of the afferent roots of the upper cervical nerves.

They examined also the influence of neck posture on the posture of the limbs in reflex standing. To do this the labyrinths were destroyed. Postures of the neck in which the head retains a symmetrical relation to the body affect the postural contraction of the right and left limbs symmetrically. Flexion of the neck, bending it ventrally, decreases the postural contraction of the extensor muscles of the fore limbs and increases that of the extensors of the hind limbs. Extension of neck lifting it dorsally increases the postural extension of the fore limbs and decreases that of the extensors of the hind limbs. Postures of the neck bringing the head out of symmetrical relation to the body influence the limb postures asymmetrically; the limbs of that side to which the lower jaw and snout are turned exhibit increased extensor action, the contralateral decreased extensor action.

The labyrinth affects not only the limb posture but also the neck posture. The head (labyrinth) posture which most supports the extensors of the limbs likewise most supports the postural contraction of the retraction of the neck, and conversely. Since the neck and head commonly alter their posture in combination their influences act usually in combination on the limbs. The strange postures assumed after labyrinthine extirpation and disease are largely traceable to abnormal postures thus imposed on the neck influencing in their turn the postures of the limbs.

The modifications shown by Magnus and de Kleijn [20] to occur in the postural reflex of decerebrate rigidity by influences coming from the labyrinths and muscular afferents of the neck are all of them confirmatory of the inference that that postural reflex is reflex standing. As mentioned above, depression of the neck such as occurs when the normal animal looks under a low shelf causes lowering of the fore quarters and the assumption of just such a modification of standing as occurs normally in that act. The raising and retraction of the head

and neck increases the extension of the fore limbs and lowers the hind quarters just as when the normal animal looks up at a high shelf. The bending of the neck and head to the right causes increased extension and advancement of the fore limb as normally occurs when the animal modifies its attitude for gazing round to the right. The increase of extension of the limbs when the preparation is inverted is also what happens in the normal animal when it is inverted; of course, the normal animal usually struggles against such an attitude being forced upon it, but, when that resistance is over, it, just as Verworn has shown in the guinea-pig, keeps the limbs extended strongly upwards, and may, by what is sometimes termed hypnosis, maintain that protective posture for a long time, even when left to itself and free from restraint.

That in the reflex standing of the decerebrate cat and dog, the postural contraction of the anti-gravity muscles which is its essential mechanism, is in the main a proprioceptive reaction whose afferent nerves are those of the anti-gravity muscles themselves is clear. As to the nervous centres involved, the following seem the main facts: In the dog after transection of the spinal cord in the hinder thoracic region, when in the course of some months, the depression of spinal function, termed spinal shock, has subsided the hind limbs are not rarely able to stand. They can bear erect the superincumbent hind-quarters, so much so that the observer placing his hand under the hind feet can find that, on lifting the hand suddenly the erect posture of the hind limbs is sufficiently strong to maintain itself, while the hinder portion of the animal is danced up and down by the hand so placed. And the spinal hind limbs will maintain their standing posture for half an hour at a time. But it is subject to sudden lapses the cause of which may not be obvious, although it is often some evident stimulus to the foot or elsewhere exciting intercurrent reflex flexion of the limb. But although spinal centres isolated from prespinal are, after the period of spinal shock has passed, able *per se* to maintain the posture at least in fair degree in cat and dog, they cannot in the period immediately following the isolating transection. Normally, some prespinal centre, or probably several centres, is adjuvant either in the sense that the postural reflex besides employing the spinal centres employs prespinal as well, or in the sense that the spinal centres which the reflex employs are kept up to the mark for that reaction by influence exerted on them by prespinal. As to where these prespinal centres lie the following can be said. Unessential for crude maintenance of the act of standing in cat and dog are the whole fore-brain and mid-brain back to and inclusive of the

posterior colliculi. Likewise the labyrinths are not necessary since they can be destroyed and the reflex posture persist. Further, the cerebellum can be removed without the posture being annulled. Evidently the prespinal centres necessary lie in the pontine region or bulb, or both, though mainly in the former.

But though the neural mechanism of the standing posture does not essentially demand for its crude performance those mid- and hind-brain regions, these do afford it assistance, and as Magnus and de Kleijn's observations prove for the octavus nerves provide for it reflex adjustment in manifold important ways. And Weed's [40] experiments indicate that the cerebellum commonly lends it a large amount of support and doubtless of refined adjustment and correlation too.

A further question is whether in *all* cases the reflex tonus or postural action of striped muscle has its source in the afferents of the posturally acting muscle itself. A departure from this rule might appear to be given by the extrinsic muscles of the eyeball. These are pre-eminently postural in their function, since the gaze is in most cases typically a posture. The third, fourth, and sixth cranial nerves are usually regarded as being purely motor nerves. But it has been somewhat recently shown that a large number of the fibres of these nerves are afferent and supply receptive nerve-endings to the recti and obliqui muscles and their tendons. The common occurrence of squint in tabes is also evidence that the postural action (tonus) of these eyeball muscles, is based on reflex action subserved by the afferents of these muscles themselves. The squint would be evidence of a hypotonia analogous to the hypotonia of tabetic limbs.

There is, however, a case in which the reflex postural action of a muscle, not a striated one, is dependent in large measure on afferents distributed not to itself. The sphincter pupillæ, like other sphincters, exhibits marked postural action, and its degree of contraction, its tonic posture, has its reflex origin largely in the retina. Here the reflex arc, as is well known, involves certain of the fibres of the optic nerve, and central stations lying in the anterior colliculus. Here the natural stimulus exciting the reflex posture is light, and the posture itself has, of course, as its *raison d'être*, the adjustment of the organ of which it is a part in regard to that very agent which is its source of stimulation. If we apply that inference to the case of the postural action of the limb muscles in, for instance, standing, the *raison d'être* of their postural action is the adjustment of the length of the muscle against a stimulus which is gravity acting in the form of mechanical tension upon the

muscles themselves. In the case, therefore, of the limb muscles and their reflex postural action in standing, we should expect the afferents of the muscles themselves to be the media of the reflex and the agents stimulating their end-organs to be mechanical strain. That brings the difficult question, what is the natural stimulus which excites the reflex postural action of the muscles in such a case as standing?

Evidently it is some state or process resident in the posturing muscles themselves. The afferent nerve-fibres gathered up in the afferent nerve of the muscle are traceable peripherally to nerve-endings, some of which are distributed to the muscle proper, others to its tendinous, aponeurotic, and fascial appurtenances. And there is evidence that the afferent nerve of a muscle contains admixed in it constituent fibres of various reflex effect. The afferent nerve-fibres of an extensor or anti-gravity muscle appear to be of two functional kinds. One kind, α, produces reflex inhibition of its own muscle and of other allied extensor muscles and reflex contraction of flexor muscles antagonistic to the muscles it inhibits. This kind usually dominates in the reflex reaction excited from the afferent nerve by faradic stimulation of moderate or considerable intensity. The other kind, β, produces reflex contraction of its own muscle, an extensor muscle; and this kind is excited by very weak or brief faradic stimulation, and even with moderately strong faradic stimuli often makes its effect seen at the onset of the reflex response. A form of electric stimulation which seems to suit this β kind better than does faradic stimulation is serial brief non-abrupt galvanic currents such as are provided by the rotating rheonome. Evidently the α kind, if it cannot excite contraction of its muscle, cannot be answerable for the knee-jerk which there is now good ground for regarding as a true and, of course, proprioceptive reflex. To the β kind, therefore, the knee-jerk can be relegated. If it is the β kind which is responsible also for the production of the postural contraction of the muscle, and the special amenity to electric stimuli of mild intensity supports this probability, that would explain the association of the knee-jerk with tonus or, as it is preferred to say here, postural contraction. The knee-jerk though not a tonic or postural reaction itself,[1] is in many circumstances a significant test for the tonicity arc, that is, for the arc on which depends the reflex postural contraction of the muscle.

A feature of the knee-jerk related to postural reflexes, very

[1] See Sternberg, M., "Die Selmenreflexe," 1893. Vienna.

familiar and constant in its occurrence in the laboratory, is the following. When the reflex excitability of postural contraction in the knee-jerk muscle is low, as it is in the first period after spinal transection, the jerk is brisk, ample, and easily obtained, and its graphic record is of very simple character. The relaxation fall of the curve is full, drops to the zero abscissa freely, and is usually followed by some pendular oscillations, subsiding more or less rapidly. The oscillations are partly due to the inertia of the limb, partly to that of the recording apparatus.

When, however, the reflex excitability of the postural contraction of the knee extensor is high, as it is after decerebration, the knee-jerk, although its ease of elicitation may not be greater, or only slightly so, than in the pure spinal state, shows in the relaxation part of its curve a feature quite different from that of the jerk in the spinal condition. It does not drop to the abscissa line but stops short of that, recording a more or less pronounced shortening of postural length of the muscle which endures often for several seconds. In fact a *shortening reaction* has appended itself to the jerk contraction. This appended increase of the postural contraction is in laboratory experience a much more reliable indication of postural excitability—hypertonus—than are the characters of the jerk proper itself. That the knee-jerk, which is now on good grounds to be regarded as a true reflex, should commonly be brisk or exaggerated in those conditions in which the postural action or "tonus" of the extensor muscle is also well marked or exaggerated, may probably be because certain of those same reflex arcs which execute the one also execute the other. But the knee-jerk itself is not a postural reaction, and is therefore not a direct sign of tonus. And the central connexions normally involved in the postural reactions of the muscle are more extensive than those required for mere exhibition of the jerk. This would explain the fact often commented on that the briskness or amplitude of the jerk does not always go hand in hand with hypertonicity.

It was shown above that in the adjustments of postural contraction, the length of the muscle may differ greatly at different times in accordance with the maintenance of different postures, but that despite these differences of postural length the tension of the muscle may be kept practically the same. The method of this local adjustment consists largely of the two proprioceptive reflexes, the "lengthening reaction" and the "shortening reaction" described above. The lengthening reaction, as there said, is obtained on elongating

the muscle by stretching it through one of its mobile attachments; and also on elongation of the muscle under relaxation by reflex inhibition. By the former method there is increased mechanical tension of the muscle, by the latter there is no increase of its mechanical tension. But the fleshy part of the muscle undergoes a similar change of form in both procedures, that is, the muscle-fibres become longer and narrower under both procedures. This would suggest that the afferent nerve-fibres concerned in the lengthening reaction have their receptor organs in the fleshy part of the muscle and not in the tendinous. The receptive organs belonging to the fleshy part of the muscle are in the main the spindles; the Golgi organs belong to the tendon.

As regards the shortening reaction, it, like the lengthening reaction, can be obtained in either of two ways—by passively approximating the points of attachment of the muscle, or by causing the muscle to shorten itself actively by reflex contraction. By the former method the muscle in the progress of its change to a shorter length undergoes no increase of tension, by the latter method it does. But the shortening reaction ensues by both methods. The tendinous portion of the muscle, although fully participating in tension changes of the muscle, participates relatively little in the form changes, these latter affecting chiefly the fleshy portion. This suggests that the afferent nerve-fibres concerned with the shortening reaction are, as in the case of the "lengthening reaction," those which have receptor organs in the muscle tissue proper, the spindles and not those connected with the tendon organs.

The abdomen is a muscular walled chamber. Kelling [16] examined the intra-abdominal pressure in the dog before and after the animal's taking of a copious meal. He found the intra-abdominal pressure altered very little after the intake of a volume of food amounting to 50 per cent. of the total contents of the abdomen in the fasting condition. Thus, after twenty-four hours' fast the water manometer showed a pressure of 2 cm. The dog then ate 300 c.c. of meat. The dog's girth at epigastrium measured then 40 cm. as against 35 cm. previously, and at penis 30 cm. as against 27 cm. On the introduction further of 100 c.c. of air into the abdomen the manometer registered 4 cm. pressure. On then killing the animal the total abdominal contents, viscera and their contents, amounted to 950 c.c.; the stomach was found to contain 380 c.c. of food. Kelling infers from his observations that the additional volume of contents must be made room for by reflex expansion of the abdominal wall—i.e., by reflex adjustment of the postural contraction of the abdominal muscles. The diaphragm being one of the muscles

limiting the abdominal chamber, it may be recalled that Dittler found that the action-currents of the diaphragm did not subside even at the end of its expiratory period; that is, a residual degree of contraction still persisted in it; this suggests a postural contraction which would contribute toward the regulation of the capacity of the abdominal chamber.

The evidence obtained from the skeletal muscles shows, therefore, that when operated reflexly they have the power of exhibiting the same tension for different degrees of active length, for they are able to counterbalance just the same extending force whatever, within a wide range, their contraction length at any given time may happen to be. We are so accustomed to regard the muscle-fibre as an elastic string that this property of exhibiting different lengths, while exhibiting one and the same degree of tension, appears contrary to our fundamental notions of muscular activity. It is a property of which the skeletal muscle, when not under the action of nerve-centres, shows no trace, and it is a property which, so far as has yet been found, is little or not at all exhibited by muscle under artificial stimulation of its motor nerve. It is, however, a property which is especially present in skeletal muscle when the nerve-centres are operating it for the maintenance of posture. It characterizes the postural contraction of skeletal muscle, and it does so in the invertebrata as well as the vertebrata. We may next inquire whether other muscles than the skeletal exhibit it; whether the visceral and vascular systems of muscle also give evidence of its occurrence in their activities.

III.—Visceral Muscle.

Bladder.—In 1882, Mosso and Pellacani [26] published observations upon the urinary bladder, in man and in the dog, difficult to harmonize with the view, then general, which regarded the viscus as simply an elastic reservoir, capable of emptying itself by muscular contraction when under accumulation of its contents it became "full." As to the manner of its evacuation their observations brought little new, but as to its being simply an elastic reservoir their observations were revolutionary. If the bladder be a flexible elastic membrane such as is a rubber bag, it must, under distension by its gathering contents, develop steadily an increasing pressure as the volume of fluid increases within it. Starting from emptiness such a bag will first unfold its flexures, and after that a rise of pressure within it must accompany the stretching of the elastic

wall. Leaving aside the hydrostatic pressure of its contents as for our purpose negligible, the bag's pressure will until its flexures are unfolded be practically zero. The zero pressure obtaining at the beginning extends therefore over a short range of capacities the upper limit of which is determined by the size to which the bag can be unfolded without being otherwise extended. For every quantity beyond this the pressure will be positive, and each increase of contents will heighten the pressure though, as experiment shows, not simply proportionally with the increased volume of the bag. The actual amount of increase of pressure for a given increase of contents will depend on the elasticity of the bag-wall.

The relation between increments of volume and of pressure depends not only on the co-efficient of elasticity of the membrane composing the bag, but on certain other factors as well. Osborne [29] has dealt with this theoretically and by experimental observation. Where, as with rubber, the extensibility of the elastic material is great, its dimensions, including its thickness, alter much under the stretch imposed. This complicates the problem of determining the co-efficient of elasticity. It was to lessen this difficulty that Fick, for whom muscular contraction was in the main a problem of elasticity, devised the isometric registration of the contraction of frog's muscle, which allows scarcely any alteration of the length of the muscle even when its tension alters greatly. In thin-walled elastic bags and hollow viscera this change of configuration of the elastic structure is large and creates serious complication for the analysis of results. A further complicating factor is that a subspherical bag may change in general figure as its size alters. And further factors still are certain obscure changes in the physical consistence of the rubber membrane as inflation and deflation proceed. An "initial rigidity" (Osborne) makes the membrane less extensible at first than later. And there is with rubber an elasticity after-action negligible with ordinary solids, which makes the rise of pressure established by an increment of distension tend to fall off and be imperfectly maintained. A time factor, therefore, enters to some extent into the relation between the bag's capacity and pressure. Further, in the bag when distended beyond a certain point the pressure instead of rising further on further distension may actually fall somewhat, though on then lessening the contents-volume there is no re-ascent of pressure. This Osborne inclines to attribute to "initial rigidity." With extreme stages of distension, when the bag is near rupturing, altered relations finally set in, but these, as wanting all likeness to conditions in the viscus, can here be left aside.

Osborne [29] compared with the behaviour of rubber bags that o the urinary bladder examined twenty-four hours after death. In this latter from its zero-pressure capacity upwards, increments of content produced somewhat rapidly increasing increments of pressure. No "initial rigidity" was found, and no point where further increase of volume was accompanied by fall in pressure. When the distension was pushed to the limit of elasticity of the wall, the gradient of ascending pressure became very steep prior to the bursting point and did not progress as a linear function of the volume. The bladder twenty-four hours after death must, we may suppose, be regarded as dead in so far as concerns the nervous and muscular constituents of its wall unless very special precautions be taken. Such a supposition is borne out by Kelling's observation on the loss of excitability of the muscular coat of the stomach within fewer hours after death. Experiments on the dead bladder would demonstrate not so much the relations obtaining between volume and pressure in the living viscus as in that viscus with a non-operative muscular coat and nerve elements. Thus they furnish a useful control for observations such as those of Mosso and Pellacani dealing, as do the latter, with the mechanical properties of the muscular wall in life.

Mosso and Pellacani [26] found in the living bladder of man and dog little evidence of a zero-pressure even in the "empty" organ, none of a zero-pressure regularly obtaining whenever the capacity was below a certain amount. In a small dog a water reservoir giving a pressure head of 5 cm. water when connected by catheter with the "emptied" bladder *in situ*, usually did not transfer any of its water to the bladder whether the latter were full or empty. The bladder was observed even at rapid intervals to hold very different volumes of water under one and the same pressure. It would exert a pressure of 10 cm. water whether its contents were 10 c.c. or 90 c.c., or a pressure of 15 cm. whether it contained 50 c.c. or 150 c.c. They concluded that in man an intravesical pressure of 18 cm. water is usually accompanied by desire to empty the viscus; and they point out that the stimulus exciting desire to micturate is closely related with intravesical pressure but not closely with the quantity of bladder content—i.e., bladder volume. They found the sensation of fulness of the bladder arise in the same person with various volumes of bladder-content according to circumstances. If, by injection of water into the bladder under a pressure of 18 to 20 cm. water, the desire to micturate were aroused, and if then the evacuation were prevented by

closing the catheter for a short time, the feeling of fulness passed off, and on re-opening the catheter the intravesical pressure was found to have subsided below the pressure at which sensation of fulness arose. The same animal's bladder might at a given time contain a larger volume of content at a lower pressure than that at which at another time it contained a smaller quantity, thus, at one time 95 c.c. at 10 cm. pressure, at another 50 c.c. at 15 cm. pressure. Speedy introduction of additional fluid into the bladder caused a temporary rise of pressure which then partly fell off, the viscus settling down to its increased contents under a pressure little above that which it had previously exerted on a much smaller quantity. In short, the viscus responded to the increment of content by a sort of active diastole proportioned to the volume of the increment. Conversely, on withdrawing from the viscus some of its content Mosso and Pellacani observed a passing fall of pressure followed usually by re-establishment of nearly the same pressure as before. The contractile bladder wall followed up the partial evacuation and then held the reduced amount with about the same light tension of grasp as it had previously exerted on the larger.

The authors remarked these unexpected features of the bladder tonus. In terms of tonus their observations were described, and would still be described, somewhat as follows. When the bladder dilated so as to hold more content its tonus was spoken of as lessened. Adhering to the literal sense tonus, however, means tension, and the salient feature of the above reaction is that the vesical tension remains little altered, and what alteration there is lies in the direction of increase. Conversely, when, after some of the viscus content has been withdrawn, the viscus wall has followed the diminution and envelops the reduced content with nearly the same, though usually a somewhat less, pressure than before, what shall be said of the change in tonus? Shall it be called more or less? Mosso, years later on a visit to England, happened to see an experiment with an isolated viscus showing change of tonus. He turned to those of us near with the exclamation, "Ah, tonus, what is tonus? Who can say what tonus is?" Either we have to get rid of the connotation "tension" from our idea of tonus, or else, as making for clearness, to avoid using the term in regard to such reactions of the viscera.

The living bladder, unlike the dead bladder or rubber bag, enfolds its contents with the same light grip whether those contents be ample or little. Similarly the hand when grasping a ball may exert a similar

light clasp whether the size of the ball be larger or smaller. The bladder sometimes enfolds a large volume with a lighter pressure than it may exert at another time on a smaller volume. In speaking of the hand clasping a handful we do not employ the word "tonus." Rather we speak of it as adopting a posture suited to the volume of the object it grasps. The case of the viscus is analogous; the bladder assumes postures suited to the volume of its contents. With 50 c.c. of content the bladder assumes what might be termed its 50 c.c. posture; with 200 c.c. contents it adopts a 200 c.c. posture. The muscular wall must in the latter case be on the average twice as long in any direction as it is the former, but such extension of it is, as shown by Mosso and Pellacani, not usually accompanied by anything more than a slight rise in its tension. The *dead* bladder has no property such that when its radius is doubled the wall-tension is hardly increased. The living muscle of the wall could exhibit such a property if, in order to double the radius, it actively relaxed from one contractile form to another, that is, if its muscular fibres actively lengthened from a shorter form which they previously maintained. But here a difficulty arises in view of the prevailing conception of muscular contraction. The notion of tension is attached as a concomitant of shortening and supposes that with greater shortening there must run greater tension. But it has become clear from recent observations, mentioned above, that skeletal muscle in postural reflex contraction may alter the length of its fibres very considerably with little or no change in the tension the muscle exerts. If we could imagine the visceral muscle of the bladder wall replaced by an appropriately shaped arrangement of living skeletal muscle acting posturally under reflex innervation it, as we have seen, could fulfil the requirements which Mosso and Pellacani's observations show that it does fulfil. We may apply the term *posture* to this property of the bladder by virtue of which it solves the problem of acting as a reservoir for quantities of fluid of very varying volume from one occasion to another without allowing the intravesical pressure to attain the reflex stimulus threshold height with one particular fluid-quantity only. But if we do, we must attempt a definition of posture in order that the term may be clear.

Posture may be passive or active. The former — for instance, the postures of a dead body impressed on it by gravity &c.—are of course outside this inquiry. Active posture largely compasses the counteraction of those effects which gravitation, &c., produce in the dead body. Active postures may be described as those *reactions*

in which the configuration of the body and of its parts is, in spite of forces tending to disturb them, preserved by the activity of contractile tissues, these tissues then functioning statically. The rôle of muscle as an executant of movements is so striking that its office in preventing movement and displacement is somewhat overlooked. When a movement, whether active or passive, has brought about a change in the configuration of a limb—e.g., by flexing one of its joints—an important function of the musculature may be to maintain the new configuration, the posture. In doing this the muscle *prevents* movement, not makes it; it then acts statically and, though in a state of contraction, does no mechanical work whether the tension it develops in thus maintaining its and the limb's new configuration be great or small. Just as the limb assumes, in result of a movement either passively imposed upon it or actively executed by it, various configurations and can maintain each of these with various degrees of tension—for instance, the arm horizontal with a 2 kgm. weight in the hand or a 6 kgm. weight—so the hollow muscular viscus after partial evacuation, or after introduction of more content, assumes a new configuration, conformably with the changed volume of content, and this new configuration is in both cases maintained by the activity of the muscle functioning statically. Here the assumption of a new configuration and the maintenance of it are muscular in the viscus just as in the limb, and the essential nature of the muscular reaction exhibited is in the viscus that which it is in the limb. Such reaction in the musculature of the limb is called postural; it is conducive to clearness if in the viscus also it be termed postural. Both are instances of the postural contraction of muscle; though the relation of the central nervous system to the postural activity is very different in the two cases. But the essential identity of the two cases justifies, and is helpfully kept in view by using, such phrases concerning a muscular reservoir viscus as that it exhibits a quarter-pint posture, or a half-pint posture, and so on, according to the amount of its contents, and that both in the one and in the other posture it may exert one and the same pressure on its contents.

The bladder is no isolated instance of a muscular viscus exhibiting these volumetric postures.

Stomach.—The musculature of this viscus is now known to function very differently in its different regions, not only in ruminants and birds, but also in the cat, dog, and man. From sphincter antri pylori to pylorus it works as a sort of mill performing forcible and more or less rhythmic movements, whereas in the fundal portion with

its contractile wall it forms a reservoir or hopper whence the mill draws its pabulum for milling. A functional requirement of the fundal reservoir is that it should receive easily and accommodate easily food arriving often in large volume quickly, and that it should do so without ensuence of any large rise of pressure. High pressure in the fundus would mean difficulty of entrance of further food from the œsophagus and the premature falling off of one of the factors essential to appetite (Cannon and Washburn [10]), if not the appearance of gastric uneasiness and pain. The mechanical problem before the fundus portion of the stomach is therefore in one way like that before the urinary bladder, it has to alter its capacity considerably without altering greatly its tension; in one respect, however, its problem is the reverse of that of the bladder, it is rapidly filled and relatively slowly emptied.

That the gastric fundus does in fact meet these requirements is shown by actual observations. Thus, in an etherized dog the stomach, exposed and freed from abdominal pressure by suitable incisions of the abdominal wall, showed during twenty minutes an intra-gastric pressure varying between 4 cm. and 5 cm. water, the intra-jejunal pressure at a distance of 40 cm. below pylorus being 2 cm. to 4 cm. water. Both stomach and jejunum at the time contained portions of a previous meal. Then 400 c.c. water were put into the stomach through a cannula. The intra-gastric pressure at once rose, but only by 1 cm. water—i.e., to 6 cm.—and twenty minutes later it had fallen to 4 cm. and the jejunal was then 3 cm. Absorption of water from the stomach is slow, especially under narcosis; nor was there evidence that the fluid had left through the pylorus. The organ had made room for the additional content and exerted on it only the same light pressure as it had done on the smaller content it had held previously. Its muscular coat had exchanged its previous 100 c.c. posture for a 500 c.c. posture practically without change of tension.

Direct observation shows the ability of the fundus to adjust suitably its postural contraction in the converse case of diminishing content. In a man with gastric fistula, by reason of a cicatricial stricture of œsophagus, Kelling obtained the following data. Water to the quantity of 200 c.c. and air to the volume of 300 c.c. were introduced into the empty stomach. The intra-gastric pressure a minute later was 10 cm. of water, but two minutes later had fallen to 8 cm., and it remained at that pressure during the next twenty minutes. It was then found that the water had disappeared from the stomach and also some of the air, 200 c.c. of the latter being all that was withdrawn.

The stomach had got rid of more than 300 c.c. of its contents without any fall in the intra-gastric pressure. The muscular coat had by a "shortening reaction," to use the term adopted above for skeletal muscle, adjusted its capacity, or, expressing the change in a linear term, shortened its contraction length without appreciably lessening or increasing its contraction tension.

The same phenomenon is exhibited by the excised surviving stomach (cat). An expansible bag, whose volume can be increased by injection from a pressure reservoir outside, is placed within the excised stomach. The intra-gastric pressure being 1·5 cm. water, the bag is then rapidly dilated within the stomach so as to raise the intra-gastric pressure to 12 cm. water. The gastric pressure soon falls to the original level again. Similarly, after a rapid injection of water the fundus portion expands (the pyloric does not) and the pressure which rises at the time of injection soon falls to what it was before the injection (Sick and Tedesco [38]). These reactions to distension the dead stomach does not give. Distension of the dead viscus to 200 c.c. causes an intra-gastric pressure higher than that attending distension to 600 c.c. capacity in the excised but still surviving organ. With the living organ *in situ*, and with its circulation intact the difference is greater still. Thus, a small cat took at a meal 150 grm. meat with 150 c.c. milk, the intra-gastric pressure rising to 9 cm. water. The animal was then killed, the stomach taken out and emptied; the introduction of 300 c.c. of fluid into it then caused the gastric pressure to rise to 80 cm. water. Cannon and Lieb [9] have shown that each passing of the cardia by swallowed food is accompanied by a rapid small dilatation of the fundus, and that this dilatation is a reflex operated through the vagus. The gastric pressure at this moment drops a little and then returns to what it had been previously.

As to the relation of the nervous system to the postural contraction which determines the capacity of a hollow muscular viscus, evidence emerges from some of the observations on the stomach. Sick and Tedesco have shown that this postural power is, to an unmistakeable extent, still retained by the excised stomach surviving in a bath of oxygenated Ringer's fluid kept at 38° C. And some observations, as yet unpublished, by Miss S. C. M. Sowton, indicate the same thing. The isolated viscus carefully shielded from changes in temperature was tested for its reactions to increments of content. The increments were introduced into the organ at its own temperature, and under pressures not much above that already obtaining in the organ at the time. After

lying locked up in the organ, by closure of the introduction tap, for a brief time, the increment of content was found to be retained in whole or in part by the organ on reopening the tap, and at a pressure often not appreciably different from that obtaining before the increment was added. But the power to adjust the postural contraction of the fundal portion to increased or diminished volumes of content without much change of pressure is, although thus present in the excised organ, less ample and less perfect than under natural conditions of the living normal organ *in situ*, with circulation and nervous system intact.

Among the invertebrata are forms which may be likened to the hollow viscera in so far that they may be regarded from our point of view as muscular bags. Sipunculus, the marine Gephyrean worm, about the size and shape of a cigar, is, so to say, such a muscular bag. Its front end lengthens to a tube, and can be extroverted and then withdrawn again, and this is a normal action when the animal bores the sand. If the hinder part, the muscular sac, be removed, tied to the lower end of a vertical glass tube filled half-way up with water, and if the glass tube and muscular sac be then plunged into a bath of sea-water, the preparation reacts as follows: The meniscus of the fluid in the glass tube settles at some point above the water-level of the bath. There is thus a certain excess of pressure in the bag over and above the fluid pressure of the bath outside it. If then the tube and preparation be plunged deeper in the bath the muscular bag becomes smaller and the height of the water-column in the glass tube still retains practically the same excess above the water-level of the bath. Conversely, if the tube and preparation be raised so that the preparation lies shallower in the bath, the size of the muscular bag increases, but the water-level in the manometric tube soon settles to practically the same height above the water-level of the bath. When the tube and preparation are raised there is, of course, a rise of hydrostatic pressure of the fluid inside the bag in reference to the water-pressure of the bath exerted on the outer face of the bag, and so conversely there is a fall when the tube and preparation are lowered farther into the bath. The muscular bag evidently actively assumes various sizes to suit the different quantities of water thus injected into or withdrawn from its interior, but it exerts practically the same pressure, maintains the same tension, whether it is holding more content or less.

IV.—Muscles of the Circulatory System.

Before leaving the unstriped musculature it may be in place to inquire what other examples of its tonus are interpretable as posture in the same sense as with the bladder and the stomach. Examination of the intestine seems to have been too little prosecuted (from this point of view) to furnish interpretable data. With the circulatory muscular apparatus, however, there are more indications. Unstriped muscle, like skeletal muscle, evidently functions for two main purposes which in some ways it is possible and desirable to consider apart. Of these one is the performance of movements which overcome resistances by the development of tension; the other is the adjustment of contractile length without necessary alteration of mechanical tension. In the muscular mechanism of the circulation these two functions seem to reach high expression in separate portions of the apparatus. In the heart the movement function, the beat contraction, predominates even more than in the bladder and stomach; and as in their case the organ is evacuated by its means. A further resemblance between heart and stomach is that just as the fundal portion of the latter is an expansible reservoir collecting provender for the strong rhythmically contracting pyloric press, so in the heart the auricles are a distensible reservoir which collects the blood, filling the strong rhythmically contracting ventricular pump. It is of the reservoir portion, the auricle, that postural function, tonus, would be expected rather than of the ventricle, and in some cases, as Fano [13] and Botazzi [6] have shown, tonus phenomena are very obvious. In the tortoise auricle, apart from beat contractions, the capacity of the chamber varies considerably at various times according to its "tonus"; and W. T. Porter [34] has drawn attention to the independence of the tonus, or state of posture as I would prefer to style it, on the one hand and of the beat contraction on the other.

In the blood-vessels as contrasted with the heart the beat contraction is but slightly developed while posturing, or "tonus" is greatly in evidence. When the fulness of an artery alters for reasons outside events in its own wall, the active posture of its circular coat should, on analogy with that of other hollow muscular organs, adjust itself to the diminished volume with little or no change in tension. The mechanical conditions of a conduit tube under a variable head of pressure, and with varying resistance in its exit, practically preclude experimental test of this in the vessel *in situ*. But the observations of McWilliam [19] and of Kesson [17] on fresh excised arteries suggest strongly that such a reaction is present. The surviving artery reveals ability to exhibit

under almost the same wall-tension considerably different diameters—i.e., circumferential lengths, capacity postures of its wall. The compensation of arterial pressure when the body is changed in position from the horizontal to the erect is, as is well known, accompanied by a change in the diameter of the arteries. This adjustment of postural capacity to a smaller content, enabling the settling down to that smaller content without appreciable diminution of their grip pressure, is analogous to the adjustment of capacity-posture in the bladder and the stomach. So also the converse reaction, when the erect position of the body is exchanged for the horizontal, finds the arteries able to meet it by postural adjustment to a new and increased postural capacity without necessarily greater tension of the arterial wall. It may be that only when the alterations of filling exceed moderate limits does the stress fall completely upon the pure elastic elements of the wall. When the arteries are examined from a freshly killed animal the arteries are found in a state of contraction. And this persists when the excised portions of them are washed out with oxygenated Locke's fluid. The excised portions are perfectly excitable to break-shock stimuli. This contracted state of the surviving arterial wall suggests that when at death the arterial tree became partially emptied by its contents running over into the venous system the postural action of the arterial wall followed down the diminishing content, just as does the bladder or stomach wall when those viscera are partially evacuated. Meyer [23] found that the best way to restore the arterial wall of the excised piece of artery to its normal calibre was to stretch it under an appropriate load for a quarter of an hour, and that in this way it resumed an approximately normal capacity and retained it after the correcting load had been removed. It will be seen that this is practically the same thing as what has in this article been described both for skeletal muscle and visceral muscle as the "lengthening reaction," although with the difference that in the case of skeletal muscle the reflex arc of the muscle, the proprioceptive arc, is necessary for the reaction, though in the case of the excised artery no such arc is required.

Bayliss [1], however, has demonstrated that the living artery reacts to a sudden rise of internal pressure by contracting, and this observation seems at variance with the above. Bayliss's reaction, I would suggest, is comparable with that contraction of a skeletal muscle which in the spinal preparation can be evoked by suddenly stretching it—e.g., by the fall of a weight attached to its tendon. The extensor of the knee, although it exhibits the lengthening reaction to a gradual

though rapid stretch, yet gives the knee-jerk to a sudden tap or quick brief pull. I cannot but think that to a more gradual distension the living artery would give a reaction of relaxation such as the bladder and stomach exhibit under similar distension. The curves given by McWilliam [19] and Kesson [17] showing the reaction of surviving arteries appear to me to support such a view.

Osborne [29] remarks, "It is a mistake to describe the flow of blood in the systemic arteries as a flow of liquid in elastic tubes. Such is certainly the case only when the muscle is fully inhibited or killed. To describe the circulation as occurring through a system of muscular tubes with some elastic tissue aiding the muscles would be more accurate." How true this is is evident from the McWilliam and Kesson experiments. The difference between the effect of adrenal extract on the excised strip of artery according as the vessel has been previously stretched or not illustrates the same point.

The upshot of the foregoing experiments on unstriped muscle is that this type of muscle, besides producing movement contractions or beats, is able to maintain itself at various lengths exerting under all those various lengths approximately one and the same tension. The wall of a hollow viscus is thus able to hold the fluid contents of the viscus at approximately the same pressure whether those contents are copious or not, because of the ability of its fibres to exert the same tension whatever the form, shorter or longer, within a certain wide range, which they have assumed. And their differences in length during this activity can be so great as to allow differences in the capaciousness of the organ suggestive of actual slipping of the muscle-fibres upon each other. Tonus as applied to such a condition seems an equivocal term. If the pressure of the wall on the viscus content be taken as criterion of tonus, then tonus has nothing to say about the state of shortness or of elongation of the muscle, for these are independent of the pressure. If tonus be transferred from its literal meaning to one descriptive of form, and be used to indicate the state of shortness of the muscle—thus, if the bladder when maintaining a small or restricted volume be considered to have more tonus than maintaining a large and "dilated" capacity—then since these states of size have no constant relation to states of tension, tonus appears a misnomer, since it then retains nothing at all of its original and literal meaning. And skeletal muscle exhibits exactly the same properties as these just described in unstriped muscle, and skeletal muscle exhibits them when it performs one of its chief functions, namely, the main-

tenance of posture. With the unstriped muscles of the viscera and blood-vessels just as with the striped muscles of the skeletal frame, it seems therefore preferable, because more direct, to speak of this form of activity as postural.

The application of the term tonus to sphincter muscles illustrates its employment as meaning posture. A sphincter is described as exhibiting tonus when it maintains a closed posture of the orifice it guards. Tonus here means nothing but postural contraction.

V.—General Remarks.

Finally, we may attempt to examine what are the main characters of the action of muscle functioning posturally.

Reflex postural contraction is characterized by (1) the low degree of tension it usually develops; (2) the long periods for which it is very commonly maintained without obvious fatigue; (3) the difficulty of obtaining by artificial—e.g., electrical stimulation—reflex contraction at all closely simulating the postural contraction produced by the natural stimuli whatever these may be; (4) by the relative ease with which reflex inhibition interrupts the postural contraction, and by the (5) "lengthening" and (6) "shortening" reactions obtainable from muscles exhibiting postural contraction. In regarding these features as distinctive various reservations have to be made. As to the first feature the tension strength, though commonly quite mild, is sometimes quite powerful. For instance, in the decerebrate cat the postural tension of the anti-gravity muscles, though usually just sufficing to support the superincumbent weight of the animal in its erect posture, may sometimes be far greater than that, both when the preparation is on its side or supine—i.e., inverted. Magnus and de Kleijn [20] have pointed out that the reinforcement of the postural extension of the limbs by influences from the labyrinths is greatest in the inverted position of the preparation. This might be taken as an indication that the reflex posture of the preparation is not really that of standing. But, as is well known, many animals when placed supine throw up the limbs into an attitude of full extension. The accentuation of the postural extension when this is done in the decerebrate preparation is, therefore, merely the reflex assumption of a defensive posture of common occurrence, and does not argue that the postural action of the extensors in the right-side-up attitude is not just as truly reflex standing.

Of more interest is the fact that when the reflex postural contraction is very powerful it seems to pass over into the full sthenic reflex action which can be produced by ordinary artificial stimulation of an afferent nerve appropriate for reflexly exciting the muscle. The lengthening reaction cannot be obtained from a muscle in ordinary sthenic reflex contraction as excited by the usual faradic stimulation of an afferent nerve; nor can this reaction be obtained, in my experience, from a posturally contracting muscle if its postural contraction is very strong. In so far then the reflex postural contraction seems when very intense to pass over into and become indistinguishable in this respect from ordinary reflex contraction such as occurs in "movement" reflexes, &c. Again, though reflex postural contraction is in many cases inhibited by stimulation of any appropriate afferent nerve with an ease and delicacy of grading truly remarkable there are instances where even powerful inhibitory stimuli fail to inhibit it. Such instances occur in certain phases of strychnine poison which augments postural contraction; also in the contraction of the fore-limb muscles in the embrace-posture of the male frog and toad at the breeding season.

It would seem, with regard to the skeletal muscles of vertebrates, that the maximal tension which the muscle can maintain posturally is distinctly less than the maximal tension which it can exert when employed for executing movements, and that when tension above that required for ordinary postures is demanded it is obtained by the same kind of contraction as that employed for movements; and that this latter kind of contraction cannot be long maintained owing to fatigue, whereas the kind of contraction maintaining ordinary postures which require less tension can be long maintained without fatigue.

The third feature characteristic of postural contraction is its relative unfatiguability. The unfatiguability is often extraordinarily great. The postural contraction of the extensors of the limbs in the decerebrate cat may persist not only for hours, but for days. I have seen it continued as far as my opportunities for observing it went, apparently without complete intermission for six days. The extremely powerful embrace-posture of the male toad continues without remission for many hours. The postural contraction of the muscle which keeps the shell of pecten shut was observed by Parnas [30] to withstand a load of 3 kilos, in addition to the pull of the elastic ligament which opens the shell, for three hours. An oyster removed from the water will keep its shell valves closed against the spring of the elastic ligament for thirty days on end. The resistance to fatigue exhibited by the muscles in cataleptic postures is well known.

It may be argued that the mild intensity of the contraction maintaining posture is the explanation of this relative immunity from fatigue exhibited by postural as compared with ordinary contraction. But, as some of the above instances show, the intensity of the postural contraction if gauged by the load which the contracted muscle can counteract is sometimes not slight.

There is another possible, and probable, reason for the unfatiguability of postural contraction. A skeletal muscle when at rest, that is, when it is not being stimulated, exhibits a certain length. It is extensile and elastic; when loaded with different weights it has under each weight, over a certain range, one definite length; as the weights are changed its length changes from that proper to one weight to that proper to another. The tensile strain of the muscle varies proportionately with the weight, so that we may say that each particular grade of tensile strain gives the muscle a particular length. When the muscle is stimulated it assumes a different and shorter form. But its tensile strain and its length are still related; over a considerable range of weights and strains the length of the contracting muscle is longer under a heavy load than under a lighter one. Further, for the muscle to assume by contraction any particular degree of shortness the excitation has to be stronger when the load is greater than when the load is less. So that tension here is the measure of the intensity of action of the muscle. Other measures of the intensity of activity of the muscle are (1) the amounts of mechanical work and heat produced by the muscle, and (2) the amount of chemical change undergone by it.

A single stimulus, that is to say, a stimulus such as a single induction shock applied to a motor nerve, which evokes a single nervous impulse as it is known to do, causes a muscle to contract with that brief contraction called technically a twitch. The usual view of the nature of any contraction lasting longer than that is that it is due to further ensuing stimuli which cause further similar twitches, and that these latter fuse with the precurrent one. The twitch is a process considerably expensive of energy; the energy appearing in the form of mechanical work and heat, and coming from chemical compounds degraded in the process of contraction. With a fused series of twitches, a tetanic contraction, the muscle usually shortens against some load till it reaches a certain grade of shortening, and this grade of shortness is then, within limits, maintained so long as the stimuli continue to be applied. Then, on withdrawal of the stimuli, the muscle lengthens under the pull of the load, returning to its original resting length. In

such a case it is, of course, only in the early part of the tetanic action—that is, when the load is actually being moved—that work in the mechanical sense is performed. When the actual moving of the load ceases, and the load is merely being retained in the new position into which it has been lifted, the muscle, although counteracting the load, is doing no work in the mechanical sense. In the former period of the tetanic contraction the muscle does external work; in the latter it does no external work. But this latter "static" period of the contraction is nevertheless expensive of muscular energy. The muscle continues to exhibit a considerable turnover of energy. It is then, it is true, playing a rôle which is merely that of an elastic band or ligament, not inexpensively, however, as to energy-cost and metabolism, as a ligament would, but at the cost of considerable energy transformations. The muscle, however, offers the advantage of supplying an elastic structure which can modify its length and elasticity according as this or that purpose is required of it. The determination of the amount of the energy-turnover in tetanised muscle thus acting statically can be estimated by measurement of the heat production or of the oxidation products. Such observations (Bernstein and Pober, Johannson) show that the energy-cost increases with the load which the contraction counteracts, and its increments increase more quickly than the increments of load. Also that the energy-cost per unit of time per kilo of load increases as the period of the contraction is prolonged. Further, in the Stockholm laboratory, it was found that the energy-cost increases rapidly with increase of the degree of shortening which the contracting muscle maintains.

As to the actual figure to which the cost in a typical example works out, Parnas, on the basis of a table of results by Johannson and Koraen, arrives at the following for the flexors of the elbow in man; 2·5 mg. CO_2 per second for a 49 kilo load, and 8·3 mg. CO_2 per second for a 160 kilo load. Such a cost accords with the view that the muscle, in thus supporting a load which it has lifted, is continuing a process essentially the same as that underlying the lifting period when external work is done; in short, that it is continuing in an additive way essentially the same process as that underlying the twitch. And it is not surprising hat such a process should entail, when prolonged, relatively rapid signs of exhaustion or fatigue.

But when we turn to inquire the energy-cost of some postures as measured by the chemical amount of oxidation products which they entail and by the less measurable but perhaps equally significant speed

of onset of signs of ensuent fatigue we find a discrepancy between the cost of these postures and that of prolonged ordinary tetanic contraction. Parnas, as the result of experiments on the postural action of the adductor of the shell of molluscs above described, shows that a weight can be supported by the muscle acting posturally with only a trifling increase of the oxygen intake or carbon dioxide output. He calculates the amount of CO_2 which would be given off by a frog's muscle holding up by tetanic contraction the same weight for the same time. He concludes that 10,000 times more energy will be expended by the frog's tetanically acting muscle than by the molluscan muscle acting posturally. According to him the experiment of Johannson and Koraen reckoned for a load of 3 kilo gives a cost of 403 mg. O_2 per hour; whereas with pecten he found where the adductor supported 3 kilo the consumption of O_2 per hour was 0·008 mg. It has to be remembered that the mass of muscle concerned in the human case was very much greater than in the case of pecten, although that in some ways only makes the contrast the more remarkable. The factors involved in the comparison are, it must be admitted, extremely difficult to assess.

H. Meyer and Alfred Fröhlich [22] noted that the enduring contraction produced by tetanus bacillus involves an extremely low metabolism, the glycogen content even increasing in the rigid muscles.

Roaf made observations on the CO_2 output of the decerebrate cat during prolonged postural rigidity of its "standing" musculature. He found the CO_2 output in this condition not detectably greater than during complete muscular paralysis by curare. Bayliss also has found the chemical turnover accompanying tonic contraction of skeletal muscles extremely low, although distinctly above that of the muscles when at rest.

The facts tend to show that in many cases postural contraction is astonishingly economically maintained; that the turnover of chemical energy involved by it is extremely low. In such cases its relative unfatiguability may well be related to its economy of maintenance. So strikingly does this aspect of it contrast with the expense of maintenance and the relatively rapid fatiguability of ordinary tetanic contraction as to suggest that the chemico-physical process underlying postural contraction is in part at least essentially other than that underlying twitch contraction, and the fusion of twitch contractions termed tetanic contraction.

The supposition has been put forward that in maintaining this economical postural contraction the muscle-fibre, or some part of it,

clots, changes from *sol* to *gel*. For instance, that with pecten, the glassy adductor, the postural adductor, when, under the approximation of its attachment points, as the valves near each other by the action of the movement muscle, it has become shorter and thicker, "clots," changes to a gel, becomes semisolid, retaining that configuration which it had at the moment when it entered into the solid phase. The posture of closure of the shell valves is then maintained, without further expenditure of energy, by the mere elasticity of this new-formed solid connexion between them. The clot or gel it must be supposed can be dissolved by nervous influence reaching the muscle. When this happens the contents of the muscle-fibres melt again and the muscle relaxes, the shell valves opening apart under the pull of their elastic ligament. On this view evidently one of the nerves supplied to the postural adductor can cause the contents of its muscle-fibres to gel, to solidify; another of its nerves can cause it to unclot.

And the postural adductor can also contract in the sense that it can shorten itself; but this shortening proceeds more sluggishly than does that of the movement adduction, whose fibres are striated.

Such a view is easily applicable to visceral muscle such as vesical and gastric, and to that of the blood-vessel wall. In extending it to vertebrate skeletal muscle points of modification would be the following:—

In them postural contraction, just like other contraction, is under natural circumstances only present in response to nervous impulses reaching the muscle from the motor neuron. A number of observations show that in postural contraction the muscle exhibits action-currents succeeding each other at rates varying with different muscles and under different circumstances from 40 per second to 80 or 90 per second. The maintenance of a gel state would, on the assumption that it underlies postural contraction, depend, therefore, on the receipt of nervous impulses, without which the particular part of the muscle-fibre which has solidified reverts to the fluid phase practically at once. And to these muscles no peripheral nerve-fibre is distributed stimulation of which unclots the muscle; the postural contraction in them can be readily suppressed by nervous action; in terms of the hypothesis they can be readily and at once unclotted by nervous action, but that is always brought about by suppressing the motor neuron discharge that is necessary for the maintenance of the gel. The resolution of the gel is therefore effected indirectly by afferent nerve-fibres, or connector nerve-fibres from other nerve-centres, which

inhibit the motor neurons which promote and maintain the gel. Also, since vertebrate skeletal muscles are obviously capable both of executing movements and maintaining postures, the question arises, in following the hypothesis, are some of the fibres of the muscle specialized for postural contraction and others for non-postural contraction? Do some solidify under nervous influence and some contract? Or do some or all of the individual fibres possess in themselves both mechanisms, a mechanism for ordinary contraction and a mechanism for resisting tensile strain by becoming semisolid? There is the theory developed by Botazzi that in the muscle-fibre the sarcostyles are the contractile mechanism, and the sarcoplasm the tonic mechanism, or, as I would prefer to say, the postural mechanism. On the view now discussed the sarcoplasm therefore would be the coagulable or solidifiable element. The two diseased conditions, congenital myotonia and myasthenia gravis, suggest forcibly that the functional mechanical activity of muscle is twofold in nature, tonic and non-tonic, postural and non-postural. Farquhar Buzzard [8] has treated of the applications of the "functional duality" theories of Grützner and Botazzi to clinical phenomena and problems, and has shown how full of suggestion such a theme can be made. And it may be recalled that Perroncito [33] (1902) asserts the presence of two kinds of motor-endings in the striped muscle, one the well-known motor-plates of medullate nerve-fibres, the other an ending belonging to non-medullate nerve-fibres reaching the muscle; and Boeke [4] confirms such a view. Mosso [25] (1905) put forward the suggestion that the latter endings are for tonic contraction and from the sympathic. De Boer has recently ascribed the Brondgeest phenomenon of the tonus of the flexors of the frog as due to severance of sympathetic fibres passing through the sciatic nerve-roots, and suggests that the cerebrospinal motor nerves control the carbohydrate metabolism, the sympathetic the protein (tonus) metabolism of the muscle. Pekelharing [31, 32] has indeed shown that prolonged tonic action of the skeletal muscles, as in the reflex "standing" of the decerebrate cat, is accompanied by an increase of creatin in muscle and creatinin output.

Even where, as in Pecten and Echinus, the muscle-cells for movement contraction and postural action are so specifically differentiated, the view does not necessarily imply that the process of contraction in them is wholly different. Though the twitch contraction of a frog's muscle isotonically recorded retains its new and shortened form hardly at all, the twitch of a tortoise muscle retains it for a fifth of a second.

In the twitch of the bladder muscle it is retained for much longer. Such authorities as Fick and Hermann have suggested analogy between the contracted state of rigor mortis, known to be due to clotting in the muscle-fibres, and the contracted state in the twitch and in tetanus contraction. Such considerations indicate that what in postural muscle is a long enduring clot phase may also be present in movement muscle though in these as a very brief phase.

Biedermann [3] (1904) wrote: "Tonus of muscle is a state of enduring shortness which only in part depends on enduring excitation by nervous centres, and for its other part is produced by those active processes in contracted muscle which otherwise cause relaxation and lengthening, being either not at all or only incompletely present to undo the alterations brought about in the muscle by the precurrent excitation." Parnas [30] (1910), after criticizing the want of clearness often attaching to the conception of tonus—he excepts, however, Biedermann's above quoted sentence—offers the following: Tonus is every stationary state of a normal muscle in which for each given length—less than the longest occurring in the unstretched muscle—the turnover of energy is independent of the load. Tonus muscles are those muscles which throughout the whole range of lengths, which extends between the longest and the shortest lengths occurring physiologically, can remain at one unaltered length both when under tension (loaded) or not under tension, and maintain that length without increasing their energy-turnover. He proposes length as a measure of tonus, thus, if L is the length of the muscle in its shortest tonic length, and l its tonic length as observed on a particular occasion, $\frac{L}{l}$ is the measure of the tonus (T) at that time. Thus if gastrocnemius be 30 mm. long at shortest and its tonic length at a given time is 35, $\frac{30}{35} = \frac{6}{7}$ T.

These definitions are of interest here because they attempt to describe a kind of activity of muscle unlike that usually accepted as the universally typical one, namely, the tetanic contraction produced by faradizing the muscle or its motor nerve. The naïve view of postural action would regard it as a mild degree of ordinary tetanic contraction. It has been the object of the present article to put forward and examine the suggestion that the kind of activity of muscle which the above definitions attempt to describe is that of muscle when functioning in posture, and that it extends not only to the posturing of the skeletal musculature but to that of the visceral and vascular musculature as well. A prime feature of this activity is that in it the length and mechanical tension of the muscle are variables

largely disconnected one from the other, and that any required shortened form of the muscle may be maintained with much greater economy of metabolism than in ordinary tetanic contraction, and with great relative freedom from fatigue.

In a previous contribution to this Journal (1906) [37] an outline was attempted of what was there termed the proprioceptive division of the nervous system. That division, it was shown, has distinctive features anatomical and functional, rendering advisable its consideration as a mechanism with peculiarities sufficiently its own to warrant its being dealt with broadly as an entity by itself. The postural action of muscles and nerves, the subject of the present paper, is a main outcome of the functioning of the proprioceptive part of the nervous system, at least it is so as regards the skeletal musculature, perhaps as regards the visceral and vascular musculature also. Reflex maintenance and adjustment of posture is a chief portion of the reflex work of the proprioceptive system, just as sensation of and perception of posture is a chief portion of the psychical output of that system.

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ON MUSCLE TONUS.

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Amsterdam.

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CHAPTER I.—THE ATONIC MUSCLE.

(*A*) *The Load Variable.*

These researches are based upon the conception of a simple proprioceptive reflex arc upon which the tonus depends. As soon as this arc is broken the muscle becomes atonic. My experiments on the atonic muscle have only been made with the M. gastrocnemius of the frog. In the beginning I cut through the motor nerve, but because it was not excluded that motor fibres might also enter the muscle with the blood-vessels, I took the muscle entirely out of the body and suspended it in a moist chamber. When the muscle is not compelled to do active work and the experiment does not last too long, the suppression of the blood circulation seems to have no sensible influence upon the elastic properties of the frog's muscle.

The units adopted in this part of the research are: 1 second as the unit of time $[\tau]$; $\frac{1}{10}$ mm. as the unit of length [L, l]. The enlargement by the lever of the length-recorder amounted to 5·65, so that the real unit of length is $\frac{1}{56{\cdot}5}$ mm.; 1 gramme as the unit of weight. The derived quantities are: The increment in length of the muscle per gramme increase of the load $\left[\frac{\Delta L}{\Delta P}\right]$. I only use this quantity under the condition that the elastic after-phenomenon is eliminated, in which case $\left[\frac{\Delta L}{\Delta P}\right]$ represents the *elasticity* of the muscle; the increment in length of the muscle per second $\left[\frac{\Delta L}{\Delta \tau}\right]$,

when the load is constant, or the influence of the variation of the load upon the value of this quantity, is eliminated. Under these conditions the quantity $\left[\frac{\Delta L}{\Delta \tau}\right]$ represents the after-lengthening or the after-shortening of the muscle. Because only plastic bodies show these after-phenomena, I propose to consider the quantity $\left[\frac{\Delta L}{\Delta \tau}\right]$ as a measure of the *plasticity* of the muscle. There are, of course, other criteria of plasticity, but the after-phenomenon seems to me the most simple and most valuable.[1]

Elasticity and plasticity are allied phenomena, but they differ in many fundamental respects. Elasticity may be defined as momentary

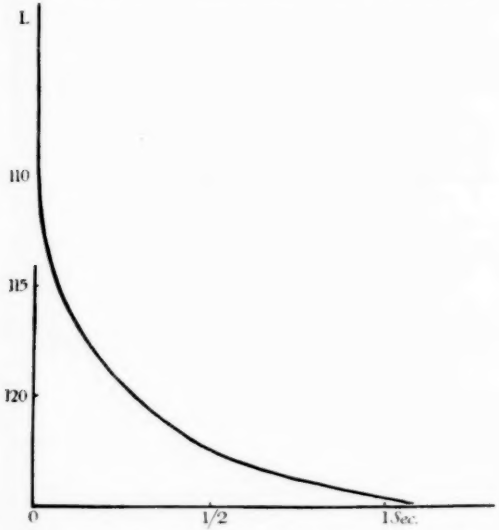

FIG. 1.—Reproduction of the beginning of the extension curve of Experiment 1.

yielding to a sudden stress, plasticity as slow yielding to a continuous stress. Elasticity is a reversible phenomenon, because the strain disappears entirely with the removal of the stress; plasticity is an irreversible one. After a prolonged stress the body does not return to its original state, but tends to a state different from the first. The variation induced in the body is therefore a permanent deformation. The property of showing definite permanent deformations under the

[1] Cf. Noyons, A. K. M., in his dissertation "Over den Autotonus der Spieren," 1908.

influence of external forces of a certain finite duration is the chief criterion of plastic bodies [38].

Experiment 1, *November* 9, 1904.—In this experiment the M. gastrocnemius was cut out of the body at 12.10 p.m., suspended in the moist chamber and charged with 2·5 grm. The length of the muscle under this load was 3·2 cm. At 12.15 p.m. this load was abruptly increased by 22 grm. In consequence of this increase of the load the muscle lengthened, but the surcharge was immediately taken away. At this moment the muscle shortened and returned to its original length. The return to the original length under these conditions was also observed by Blix [1]. The elasticity of the muscle amounted in this

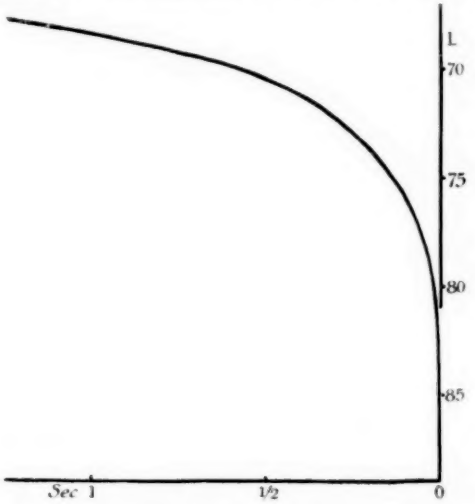

FIG. 2.—Reproduction of the beginning of the unloading curve of Experiment 1.

case to 4·55. Again the charge of the muscle was increased by 22 grm., but now this surcharge was left on the muscle for $51\frac{3}{4}$ minutes. Then the surcharge was taken away. Table I gives the result of this experiment. After a sudden elongation of about 109 units, the muscle continued to lengthen, and after $51\frac{3}{4}$ minutes this after-lengthening had not finished. Figure 1 is the reproduction of the beginning of the tracing, and we see that the initial elongation passes continuously into the after-lengthening. For this reason it is not possible to determine with accuracy the elastic elongation, and hence I choose the time-notation $0 + \epsilon$, ϵ signifying a short, not quite determined interval of time. For

the same reason a mark of interrogation is placed behind the first number (109) in the second row. This notation indicates that in a short interval of time (ϵ) an elongation takes place of 109 units, and that this elongation is approximately the elastic lengthening of the muscle under the influence of the surcharge of 22 grm. Fig. 2 reproduces the unloading of the muscle from the surcharge, and we see that here also the sudden shortening continuously passes into the after-shortening.

In the third row of Table I, I give under $\left[\frac{\Delta L}{\Delta \tau}\right]$ the plasticity of the muscle. We notice that under the influence of the surcharge the plasticity diminishes, but even after 51 minutes the after-lengthening is still going on. The same thing happens after the unloading of the muscle. In Tables II and III, two other experiments of the same kind are reproduced. We conclude from these experiments *that an atonic muscle, loaded with a moderate weight (20 to 30 grm.), does not reach a definite length even in about one hour, but continues lengthening and tends finally to a state of rest.* This state of rest, as I will show later, is in general only a state of apparent equilibrium. It ensues, therefore, from this simple experiment that the length of the atonic muscle is not entirely determined by the load. The experiment proves further that after the surcharge is taken away the muscle returns to a length greater than before.

Table II contains for this purpose an extrapolation, which shows that even five and a half hours after the unloading the muscle would not have recovered its original length. We conclude from this observation *that a continued pull exercised by a moderate weight suspended on the tendon of a muscle produces in that muscle a permanent deformation.*[1]

In the fourth row I give the quantity $\left[\frac{\Delta L}{\Delta \tau} \times \tau\right]$, i.e., the plasticity multiplied into the time elapsed since the moment of loading or unloading. Within the limits of experimental error this quantity is constant. We conclude from this fact *that the after-lengthening and the after-shortening are directly proportionate to the logarithm of the time, if we take as zero of time the moment immediately preceding the loading or unloading of the atonic muscle.* I found this relation independently of the work of Percy Phillips [37], who some years earlier discovered the same rule to hold good for the after-phenomenon in soft metal wires.

[1] Mosso arrived at a similar result: "Les muscles sont comme le plomb et le beurre qui, lorsqu'ils ont été déformés, conservent indéfiniment l'empreinte qu'ils ont reçu" [33].

Experiment 4, *November* 15, 1904. — In this experiment the M. gastrocnemius was taken out of the body at 10.10 a.m., and suspended in the moist chamber at 10.15 a.m., loaded with 13 grm. Under these conditions the length of the muscle was 3·2 cm.; the experiment began at 10.32 a.m. The régime of this experiment is the following: A light weight-pan of about 13 grm. was fastened to the muscle; in this pan mercury may flow in and out at a constant rate, and in this way a regular rising and falling of the load was attained.

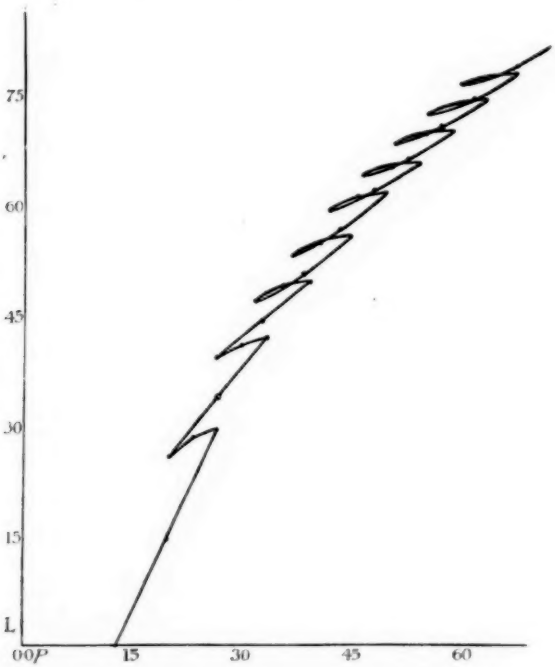

FIG. 3.—Length-load diagram of Experiment 4.

In the third row of Table IV the rate of increase and decrease of the weight is given in grammes per minute. During the course of the experiment the rate of increase, as well as the rate of decrease, varied a little, but these deviations from constancy do not visibly influence the form and the direction of the curves. The duration of the inflow is six minutes, and that of the outflow is three minutes. The total period of nine minutes is usually called a cycle, and the variation of the load

during that interval of time a cyclical variation. Because the outflow does not exactly compensate the inflow, there is at the end of each cycle a surplus. In that case we speak of non-closed cycles. The régime is therefore one of non-closed cyclical variations of the load.

Fig. 3 is the graphic representation of this experiment. Each cycle is composed of a rising and a falling load curve, agreeing respectively with a phase of increasing and decreasing load. As a general result we find that the falling curve lies above the rising curve of the same cycle. The meaning of this result is that the increasing load has not only provoked an elastic elongation, but also a plastic deformation of the muscle. The same fact we notice if we compare a falling curve with a rising one of the next cycle. These two curves have only one common point, viz., the first two cycles or the curves form a loop. If the phenomenon was only an elastic one, it would be reversible, and the two curves should agree. Hence the formation of a loop proves that there is also an irreversible part in the phenomenon, and this irreversible part is the plastic deformation of the muscle, caused by the cyclical variation of the load.

Experiment 5, *November* 16, 1904, is of the same kind (Table V, fig. 4). It shows that even after twenty cyclical variations of the load the plastic deformation is still going on. We conclude, therefore, *that a cyclical variation of the load produces in the atonic muscle a deformation, and that this deformation, according to its plastic nature, is at least partly a permanent one.*

We may arrive at the same conclusion in a somewhat different way. If we draw a vertical line in figs. 3 and 4, this line, if it cuts the tracing at all, cuts the curve generally in more than one point; the same happens if we draw a horizontal line. Because along a vertical line the load is constant, and along a horizontal line the length is invariable, we conclude *that the length of the atonic muscle is not entirely determined by the load, and inversely that a certain load does not necessarily agree with a fixed length.* In other words, the length of the muscle is partly determined by the plastic deformation produced by the load, and the amount of this deformation depends upon the way the load reached its actual value. Systems of this kind are usually called systems endowed with hysteresis, and the phenomenon, that the state of these systems is not entirely limited by external forces, is called hysteresis.

Fig. 4 presents another feature of some interest. It shows that the loops, from nearly vertical in the beginning, take on a more and

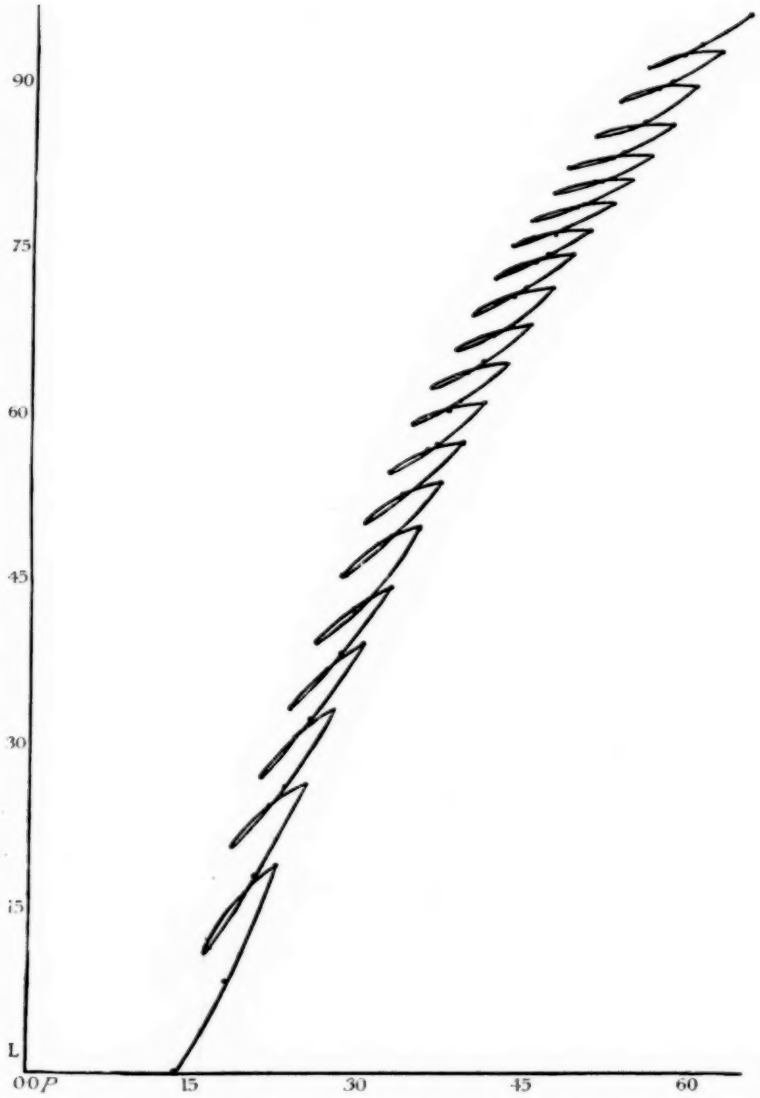

FIG. 4.—Length-load diagram of Experiment 5.

more horizontal position in the end. This means that the plasticity of the muscle diminishes in the course of the experiment.

The régime of Experiments 6 and 7 consists in augmenting the charge of the muscle per saltim.

Experiment 6, *December* 30, 1904 (Table VI).—The muscle was cut out at 10.15 a.m., brought into the moist chamber at 10.23 a.m. and loaded with an initial charge of 4·8 grm. At 10.25 a.m. this load was

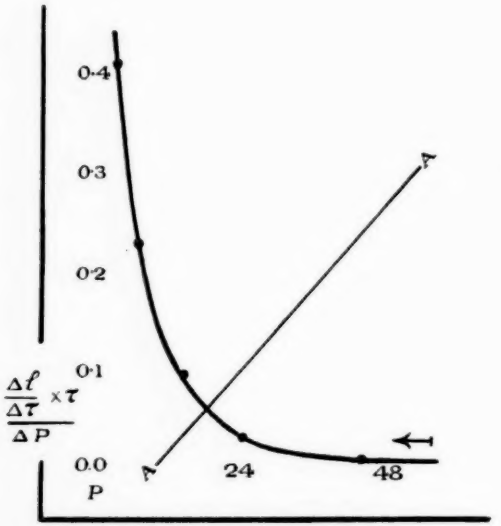

FIG. 5.—Graphic representation of the results of Experiment 6. The reduced constant of plasticity as a function of the charge of the muscle during each period of after-shortening.

increased with 3, 7, 10, 20 and 30 grm. at intervals of one minute. Then I observed the after-lengthening for about an hour, and found that the mean value of the constant $\left[\frac{\Delta L}{\Delta \tau} \times \tau\right]$ was 7·27. I propose to call this quantity the *constant of plasticity*, because its value depends upon the value of $\left[\frac{\Delta L}{\Delta \tau}\right]$. An hour after the beginning of the experiment the weight of 30 grm. was lifted. The mean value of the constant of plasticity, deduced from the after-shortening of the muscle during the next half hour, is 0·39. Then the weight of 20 grm. was taken away, and so on, always with intervals of about half an hour. Table VII contains the values of the constant of plasticity after each successive

lifting of the load. It follows from this table that the load lifted influences the value of this constant. In order to demonstrate this influence, I divided the mean value of the constant of plasticity in each interval succeeding the lifting of a weight by the value of the weight taken away. The fourth row of Table VII $\left[\frac{\frac{\Delta L}{\Delta \tau} \times \tau}{\Delta P}\right]$

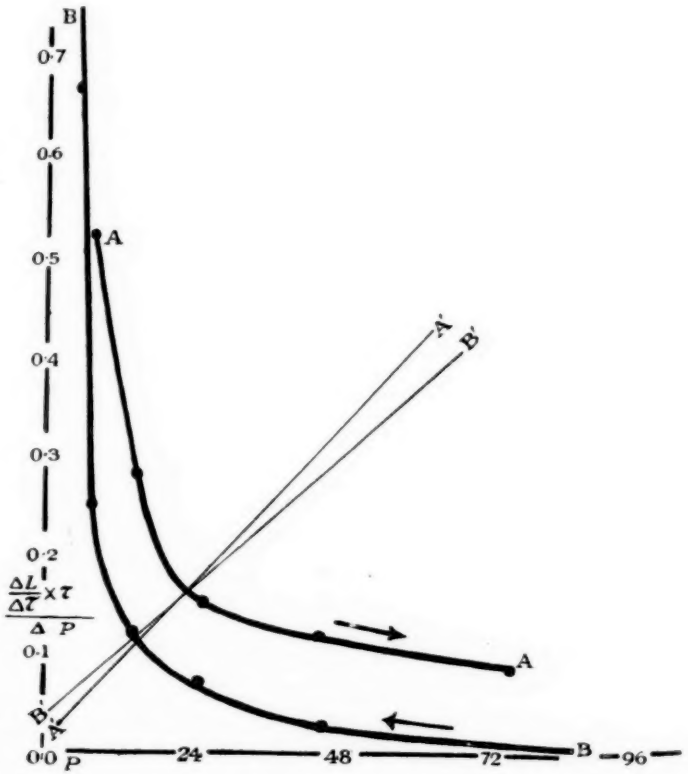

FIG. 6.—Graphic representation of the results of Experiment 7. The reduced constant of plasticity as a function of the charge of the muscle during each period of after-lengthening (AA curve), and during each period of after-shortening (BB curve).

gives these values. In fig. 5 I plotted the value of this constant against the charge of the muscle during each period of after-shortening. The curve which ensues is symmetrical in respect to the axis AA and resembles a hyperbola.

Experiment 7 (Tables VIII, IX) is of the same kind, but in that case I observed the after-lengthening of the muscle after each increment of the load, as well as the after-shortening after each successive lift. Fig. 6 is the graphic representation of the results of the experiments. The arrows along the curves show the direction in which the load was varied. Each of the two curves is symmetrical, but the axis of symmetry of the two tracings do not coincide. Both curves resemble a hyperbola. The curve AA, corresponding to the successive loading of the muscle, lies above the curve of the discharging (BB), which proves that the charging and discharging per saltim of the muscle also produces in the muscle a plastic deformation. The more special conclusions which might be drawn from these experiments are of physical rather than of physiological interest. I conclude, therefore, only *that the value of the constant of plasticity depends as much upon the charge of the muscle as a whole as upon the load which is added or lifted.*

The régime of Experiments 8 and 9 consists in successive series of cyclical variations of the load.

Experiment 8, *November* 30, 1904 (Table X).—The muscle was taken out of the body at 12.40 p.m., suspended in the moist chamber, and charged with 3·65 grm. The length of the muscle under this initial charge was 3 cm. At 1.11 p.m. the load was augmented with 10 grm. The lengthening of the muscle was observed for about twenty minutes. The mean value of the constant of plasticity during that interval amounted to 4·12. Now the weight of 10 grm. was carefully, and without shocks, removed from the weight-pan and immediately replaced in the same way. This procedure means a quick variation of the load from $(3{\cdot}65 + 10) \rightarrow 3{\cdot}65$, and again from $3{\cdot}65 \rightarrow (3{\cdot}65 + 10)$. This cyclical variation of the weight was repeated five times in succession; the time necessary for the performance of these five cyclical variations of the load was about one minute. By these five cyclical variations the after-lengthening is set up, but this effect quickly dies away. If we take as the zero of time the moment immediately before the last cyclical variation was terminated, we find for the constant of plasticity the mean value 1·19. A new series of five cyclical variations again activated the after-phenomenon, but the constant of plasticity is only 0·75. The general result is now that each series of cyclical variations of the load exalts the after-lengthening for a short while, but that the constant of plasticity decreases regularly after each series.

Fig. 7 is the graphic representation of Experiment 8. Along the

abscissa I placed the ordinal number of the series of cyclical variations; 1 means, therefore, the first series; 2 the second series of five cyclical variations of the load, and so on. Because the cyclical variations were

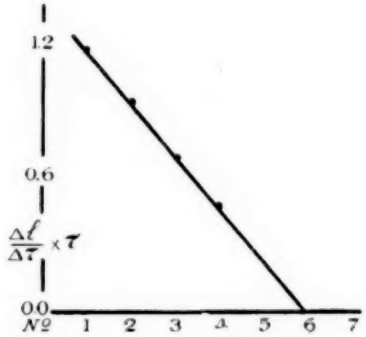

FIG. 7.—Graphic representation of the result of Experiment 8. The constant of plasticity as a function of the ordinal number of the series of cyclical variations of the load.

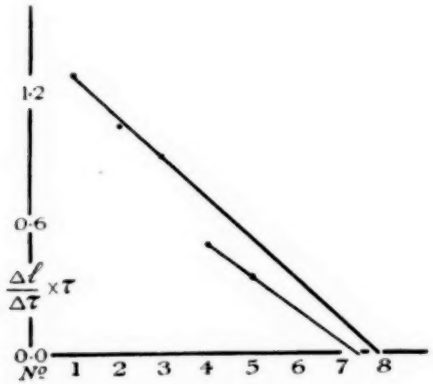

FIG. 8.—Graphic representation of the result of Experiment 9. The constant of plasticity as a function of the ordinal number of the series of cyclical variations of the load.

repeated with equal intervals of time, I placed these numbers equidistant. The ordinates represent the mean value of the constant of plasticity during the interval following a set of five cyclical variations. The curve which ensues is rectilinear. The experiment shows, therefore,

that the effect of a series of cyclical variations of the load quickly diminishes, and that probably the effect of the sixth or seventh series would be *nil*. Successive series of cyclical variations, therefore, lead to a state of rest. The next experiment will prove that this state of rest is not a state of true equilibrium.

Experiment 9 (Table XI) is of the same kind. The surcharge amounted in this experiment to 30 grm. The régime was not uniform throughout, but after three series each of five cyclical variations followed two other series of ten cyclical variations. Fig. 8 reproduces the result of the experiment in the same manner as fig. 7. The five points representing the five series of cyclical variations do not lie upon the same straight line, but belong to two different lines. The effect of a series of cyclical variations, therefore, depends not only upon the weight which is varied, but also upon the number of cyclical variations of the weight. The lines converge, but do not cut the abscissa in the same point. The final state reached through successive series of cyclical variations of the same load, therefore, are not states of true equilibrium. If this were the case, the lines ought to converge to a common point of the abscissa. We conclude, therefore, *that the final state reached by an atonic muscle under the influence of a constant load is only a state of apparent equilibrium, and that under the influence of successive cyclical variations of that load the muscle approaches slowly to a state of true equilibrium.* The length of the muscle in a state of true equilibrium probably equals the *post-mortem* length.

The régime of Experiment 10 (Table XII) is one of vibration. I regret to have performed only one experiment of this kind. The reason why I did so is, that at the moment I performed these experiments, I had but a very dim notion of what would ensue. The frog used for the experiment was killed at 10.50 a.m., the muscle cut out at 1.10 p.m., suspended in the moist chamber at 1.20, and loaded with 3·1 grm. At 1.30 p.m. this charge was abruptly increased with 20 grm. I observed the after-lengthening for ten minutes. The mean value of the constant of plasticity during that interval amounted to 3·25. With the aid of this constant, the continuous line representing $\left[\frac{\Delta L}{\Delta \tau}\right]$ as a function of the time $[\tau]$ is extrapolated (fig. 9). At 1.40 p.m. the muscle was thrown into longitudinal vibration; the duration of each period of vibration was about 1·5 minutes, but because the vibration slowly died away, this period was not entirely fixed. With intervals of ten minutes, the vibration was repeated. In each successive period the amplitude of the movement, as well as its duration, were slightly

increased. This increment, however, was very considerable for the last period. The load was constant during the experiment, and not lifted; hence it is possible to determine the after-elongation during the period of vibration. The broken line in fig. 9 represents the effect of the vibrations upon the value of the plasticity. In reality the line will be fluent, and not broken, but my determinations of the value of $\left[\frac{\Delta L}{\Delta \tau}\right]$ are too scanty to fix more exactly the character of the line. Zigzag lines indicate in

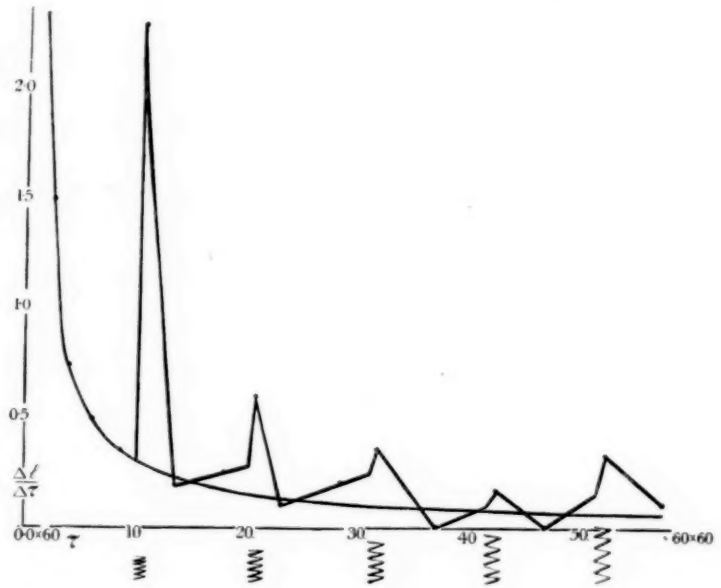

FIG. 9.—Graphic representation of the effect of vibrations upon the value of the plasticity.

fig. 9 the moment the muscle was put into vibration; fig. 10 is the reproduction of the graphic records of the second and of the fifth groups of vibration.

The experiment proves that the muscle is extremely susceptible to vibrations, especially at the beginning of the after-lengthening, and that the effect of each period of vibration quickly diminishes. Only the last set of vibrations is followed by a somewhat greater effect. This increment of the effect, however, is caused by a strong increase of the amplitude and of the duration of the vibrations. This experiment is of

interest, because it elucidates the cases in which I found at some given moment a constant of plasticity larger than would agree with the relation $\left[\frac{\Delta L}{\Delta \tau} \times \tau = \text{const.}\right]$. A close examination of the tracings in these cases always showed small vibrations. In the tables these vibrations are indicated by a zigzag line. The deviations here and there from the simple logarithmic relation are therefore caused by vibrations. We conclude from Experiment 10, and from the deviations in the experiments already mentioned, *that the most efficacious expedient, to set up the after-lengthening and to reduce the atonic muscle to its ultimate equilibrium-length, is to put the muscle into longitudinal vibration.* The weight with which the muscle is loaded probably influences the course of this process, in so far that the final length is more quickly reached with a moderate load than with a light load.

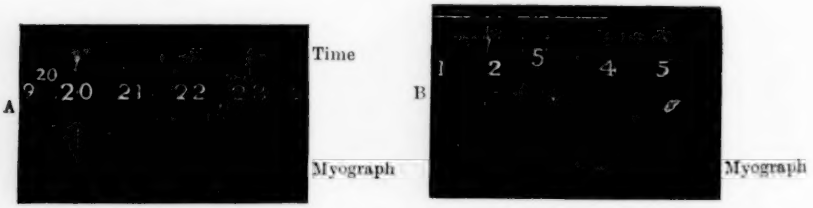

FIG. 10, A and B.—The effect of longitudinal vibrations upon the length of the atonic muscle. Fig. A is the reproduction of the graphic record of the second group of vibrations, and fig. B is the reproduction of the fifth group of vibrations. The enlargement is about ten times.

The phenomena so far described only hold good for moderate charges. The upper limit for the frog's gastrocnemius lies in the neighbourhood of about 70 grm. To prove this I reproduce the beginning of Experiment 11, December 29, 1904 (Table XIII). The muscle was charged per saltim with 74·8 grm. In this case the quantity $\left[\frac{\Delta L}{\Delta \tau} \times \tau\right]$ was not constant but increased with the time. This means that the after-lengthening did not proceed according to the logarithmic rule, but more rapidly. The same phenomenon is shown by soft metallic wires, and in these cases the constant weight ends by rupturing the wire.[1]

In the next group of experiments I have tried to eliminate the

[1] For the analogy between the atonic muscle and soft metal wires see A. Morley and G. A. Tomlinson, *Philos. Mag.*, 1906, sixth series, vol. xi, p. 380; and E. Lenoble, "Thèses de Bordeaux," 1900, No. 12.

after-phenomena. The methods adopted for this purpose are increasing and decreasing the load as slowly as possible, or varying the load very rapidly and repeating this procedure several times. In both cases the curves obtained are extension curves of the atonic muscle, and the difference lies only in the rate of increase or decrease of the weight.

The régime of *Experiment* 12, *October* 31, 1904 (Table XIV) consisted in increasing the weight as slowly as possible. In the third row of Table XIV under $\left[\frac{\Delta P}{\Delta \tau}\right]$ the increase of the load per second is given. I tried to establish a uniform rate, but, as will be seen from the table, this aim was not reached. The rate of increase of the load fell from 14 mg. to 4 mg. per second and then rose a little to 7 mg. The influence of this deviation from the uniform rate upon the form of the tracing is probably not very considerable, because it took place smoothly and without shocks. After two hours the maximum charge of 72 grm. was attained, and from that moment the load was decreased. Very soon, however, the experiment was stopped, in consequence of a defect in the apparatus.

The chief result of this experiment is, that the extension curve of the atonic muscle is nearly rectilineal, i.e., that the increase in length of the muscle is approximately proportionate to the increment of the charge. The deviation from the simple law of proportionality of strain to stress resides in the beginning of the curve and disappears towards the end. Other experiments of the same group prove that the deviation of the extension curve from the straight line becomes more and more considerable by augmenting the rate of increase of the load. We conclude, therefore, *that the extension curve of the atonic muscle is probably rectilineal when the rate of increase of the load is very small, or more exactly, that the extension curve approaches to the straight line when the rate of increase of the load approaches the zero.*

The elasticity of the muscle determined from the beginning of the extension curve is 1·08. The determination of this quantity is approximately possible, because in the beginning, when the load is very small, the after-elongation vanishes against the elastic elongation. It is also possible to determine the elasticity of the muscle from the end of the tracings, which is strictly rectilineal. The elasticity determined from that part of the curve amounts to 1·06, being the same as at the beginning, within the limits of experimental error. I conclude from this agreement *that the elasticity of the muscle is constant and that the deviation from the straight line in the beginning of the curve is caused by the after-phenomenon.* The sixth row of Table XIV gives

the plasticity of the muscle, calculated upon this base. We see that under the given régime the plasticity of the muscle is very small in comparison with the elasticity. In fig. 11 I plotted the plasticity against the time. The figure shows that at first the plasticity quickly increases, reaches a maximum, and then decreases.[1]

Experiment 13 (Table XV) is of the same kind. The mean increase of the load, however, was larger, viz., 27 mg. per second. The deviation from the mean was not so considerable as in Experiment 12. After three-quarters of an hour the maximum charge was reached, and from that moment on the load decreased. The rate of decrease of the

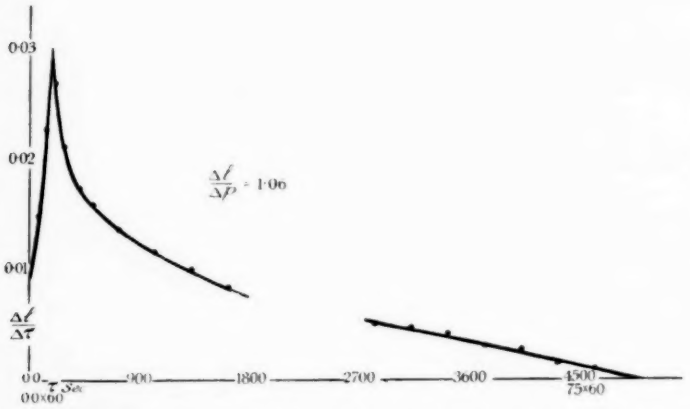

FIG. 11.—Graphic representation of the result of Experiment 12. The plasticity as a function of the time.

load was fairly constant in the beginning, but slowly diminished towards the end of the experiment. Eighty-six minutes after the commencement of the experiment the muscle was again under its initial charge. From that moment onwards I observed the after-shortening of the muscle for about thirty minutes, and found that the relation expressed by the formula $\left[\frac{\Delta L}{\Delta \tau} \times \tau = \text{const.}\right]$ is not satisfied. In this case the value of the constant first increases and then probably decreases. I found the same result in other experiments, so *that the relation* $\left[\frac{\Delta L}{\Delta \tau} \times \tau = \text{const.}\right]$ *only holds good when the time required for the loading and the unloading of the muscle is small in comparison with the time necessary for the whole*

[1] The gap in the curve is caused by a slight discontinuity in the original tracing.

cyclical variation; in other words, when the charging and discharging of the muscle takes place fairly quickly.

The elasticity determined from the beginning and from the end of the extension curve amounts to 1·25. Fig. 12 represents graphically the plasticity of the muscle. This curve resembles closely the curve of fig. 11, but from the scale upon which the curve is drawn it follows that the plasticity is about double in this case. The unloading curve is in many respects similar to the extension curve. The elasticity, determined from the beginning of the unloading curve, amounts to but 0·18. This fact proves that the muscle has undergone a plastic deformation. The

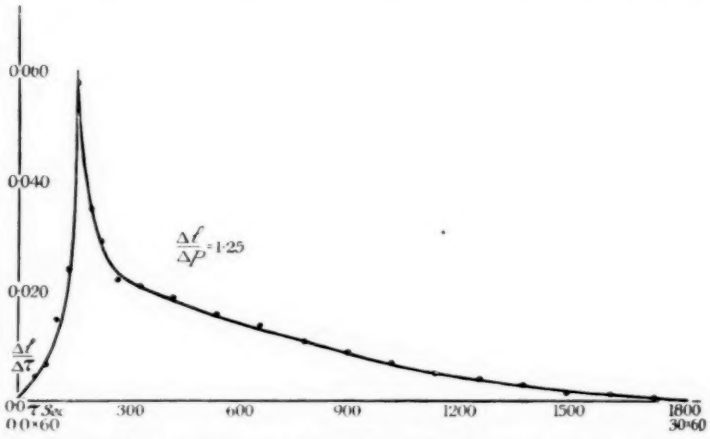

FIG. 12.—Graphic representation of the result of Experiment 13. The plasticity as a function of the time during the loading of the muscle.

plasticity curve (fig. 13), derived from the falling-load curve, resembles the plasticity curve of the rising-load tracing, but inverse. The arrow at the abscissa indicates the moment when the muscle is again under its initial charge. From that moment on the plasticity falls and then slowly slopes towards zero. Half an hour after the accomplishment of the cycle there is still an elongation of thirty-seven units.

In the three last experiments the increase of the load took place quickly and uniformly, the rate of increase of the charge varying from about 3 to 0·6 grm. per second in the successive experiments. The unloading happened abruptly and in a somewhat irregular manner, so that the period immediately following the unloading was not suitable for measurement. The zero of time necessary for the determination of the

constant of plasticity is therefore a little uncertain, and also the length of the muscle immediately after the unloading.

Experiment 14, *December* 20, 1904.—The muscle was cut out at 10·05 a.m., and suspended in the moist chamber at 10·12 a.m. under an initial charge of 10·75 grm. Some minutes later the experiment commenced; it consisted of eleven cyclical variations of the load with equal intervals of about ten minutes. The rate of increase of the load was approximately 3 grm. per second, and the charging of the muscle took nearly twenty-five seconds. The muscle was left for about five

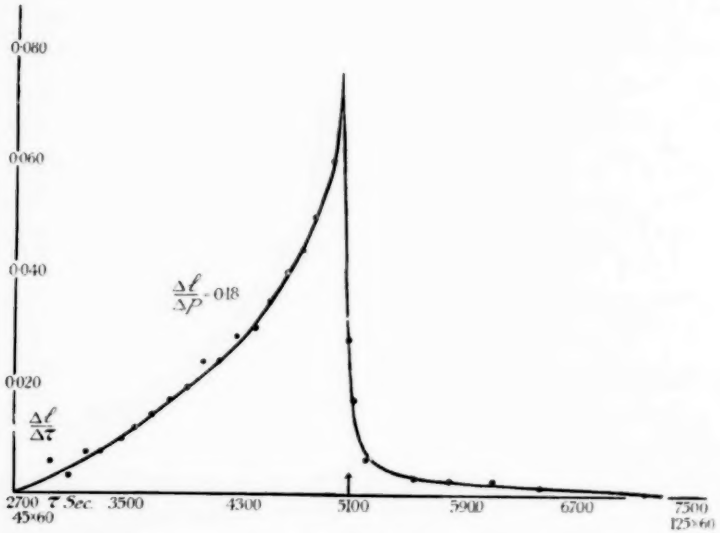

FIG. 13.—Graphic representation of the result of Experiment 13. The plasticity as a function of the time during the unloading of the muscle.

seconds under the influence of the maximum charge and then abruptly unloaded.

At the moment when the muscle was under full and constant charge we may determine the amount of the after-lengthening per second, because under these conditions the increase in length of the muscle is only due to the after-phenomenon. At the moment just before the maximum charge was attained we may determine the increment in length of the muscle provoked by the joint effect of the after-phenomenon and the increment of the charge. The difference between these two quan-

tities gives approximately the pure elastic strain of the muscle at the end of the extension curve. The elasticity of the muscle may also be determined from the beginning of the extension curve, when the increment of the load is small, and the after-lengthening therefore vanishes against the elastic elongation. In these experiments, too, the elasticity of the muscle, determined from the beginning and from the end of the tracing, is always the same within the limits of experimental error. We conclude, therefore, *that in these experiments also, where the extension curve deviates considerably from the straight line, the deviation is caused by the after-phenomenon.* The determination of the plasticity is based on this conclusion.

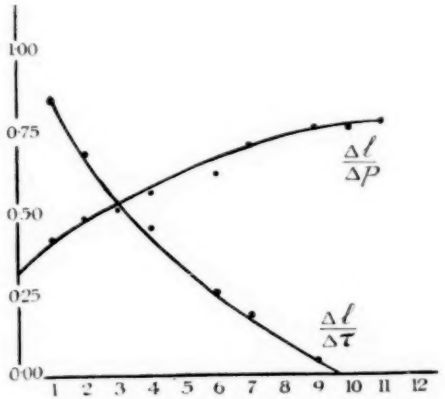

FIG. 14.—Graphic representation of the result of Experiment 14. The curve at the end of which the notation $\frac{\Delta L}{\Delta P}$ is placed represents the elasticity of the muscle in the successive cycles. The curve at the end of which the notation $\frac{\Delta L}{\Delta \tau}$ is placed represents the plasticity of the muscle at the moment of the maximum charge.

Tables XVI, XVII, and XVIII contain the measurements of the first, the sixth, and the tenth curves. Of the other curves of this series I give only the numerical results combined in Table XIX. The first row of this table contains the time necessary for the loading of the muscle in seconds; the second row the rate of increase of the load in grammes per second; the third row the elasticity of the muscle; and the fourth row the constant of plasticity during the period of after-shortening. It is evident from this table that the régime was fairly the same in the eleven

cycles. The variations in the elasticity and in the plasticity of the muscle in the course of the experiment are therefore only due to the repetition of the cyclical variation of the load. In figs. 14 and 15, the results are plotted. The numbers along the abscissa denote the successive cycles. These numbers are placed equidistant because the cycles succeeded each other with equal intervals of time. The first curve (fig. 14), at the end of which the notation $\frac{\Delta L}{\Delta P}$ is placed, represents the variation of the elasticity of the muscle in the successive cycles. We conclude from this graph *that the elasticity of the muscle is increased by repeating the*

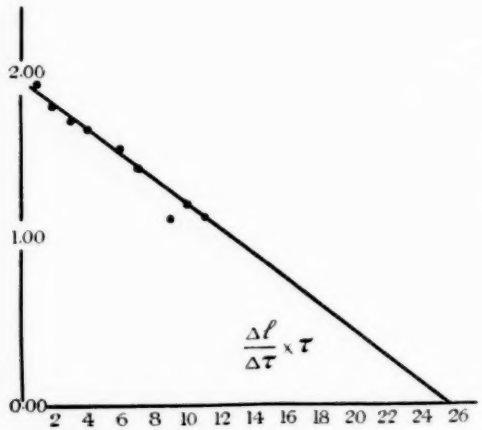

FIG. 15.—Graphic representation of the result of Experiment 14. The constant of plasticity during each interval succeeding a cyclical variation of the load. The numbers along the abscissa denote the successive cycles.

cyclical variation of the load. It is evident from the form of the curve that this quantity approaches an upper limit. A rough extrapolation makes it probable that this limit lies in the neighbourhood of 1, and that this limit would be attained after about 25 to 30 cycles. The second curve, marked $\left[\frac{\Delta L}{\Delta \tau}\right]$, represents the plasticity of the muscle exactly at the moment when the maximum charge is arrived at. The meaning of this curve is to show that the area over which the after-elongation extends narrows successively. Fig. 15 represents the constant of plasticity during each interval succeeding a cyclical variation of the load. The points representing this quantity are all grouped along a straight line, as already found (Experiments 8 and 9). By extrapolating the

line it would cut the abscissa in the neighbourhood of Number 26. This means that after 26 cycles the muscle immediately after the unloading would reach its initial length. When this is the case, the after-phenomenon is eliminated. This extrapolation tends, therefore, to the same conclusion as the extrapolation of the curve of fig. 14, viz., *that by repeating a rapid cyclical variation of the load the elastic after-phenomenon is reduced in extent and in magnitude, and that probably 25 to 30 cycles would suffice to eliminate the after-phenomenon.* At that moment the elasticity is maximum and the muscle reduced to a purely elastic body. Brodie [13] arrived in a somewhat different way at a similar result. Fig. 16 is the reproduction of the plasticity,

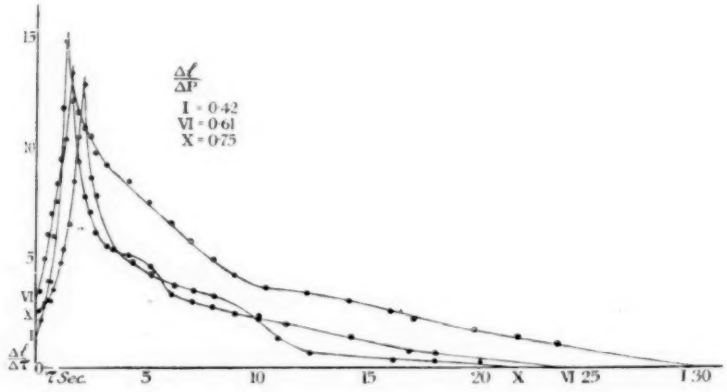

FIG. 16.—Graphic representation of the results of Experiment 14. The plasticity as a function of the time during the loading of the muscle—first, sixth and tenth extension curves.

deduced from the first, sixth and tenth extension curves. The tracings exhibit the same feature as those of figs. 11 and 12. A comparison, however, of the scales shows that the plasticity is in this case about 500 times larger than in Experiment 11, and that the time needed for the full development of the after-phenomenon is much smaller.

Experiment 15 is of the same nature as Experiment 14, but the rate of increase of the load is somewhat smaller. Table XX contains the chief results, and figs. 17 and 18, are the graphs plotted in the same way as figs. 14 and 15. The extrapolation of the curve marked $\left[\frac{\Delta L}{\Delta P}\right]$ of fig. 17 tends to the conclusion that the upper limit to which the elasticity approaches lies in the neighbourhood of 0·75, and would

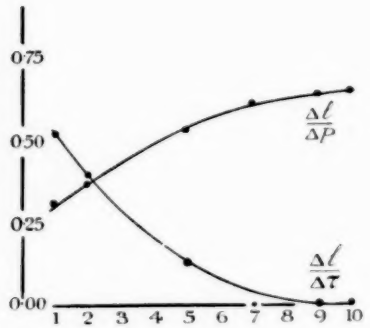

FIG. 17.—Graphic representation of the result of Experiment 15. The curve at the end of which the notation $\frac{\Delta L}{\Delta P}$ is placed represents the elasticity of the muscle in the successive cycles. The curve at the end of which the notation $\frac{\Delta L}{\Delta \tau}$ is placed represents the plasticity of the muscle at the moment of the maximum charge.

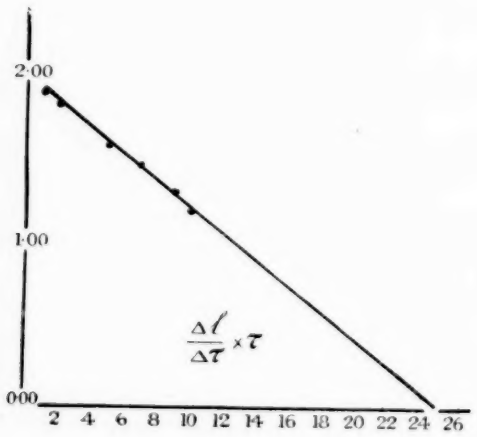

FIG. 18.—Graphic representation of the result of Experiment 15. The constant of plasticity during each interval succeeding a cyclical variation of the load. The numbers along the abscissa denote the successive cycles.

be reached after 20 to 30 cycles; the extrapolation of the straight line of fig. 18 tends to the same conclusion.

Experiment 16 is the last of this group. In this case the rate of increase of the load is still smaller, about 0·65 grm. per second. In Table XXI the results are combined; figs. 19 and 20 are the graphs. Extrapolation of the $\left[\frac{\Delta L}{\Delta P}\right]$ curve proves that the upper limit of the elasticity lies probably between 0·75 and 1, and would be reached after 20 to 25 cycles. The extrapolation of the straight line of fig. 20 is in harmony with this. Fig. 21 is the reproduction of the plasticity curves, deduced from the first, third and eighth extension curves; these curves resemble closely those of fig. 16, but the scales are different.

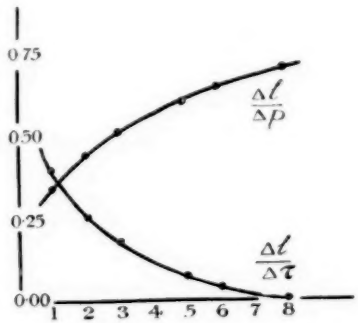

FIG. 19.—Graphic representation of the result of Experiment 16. The curve at the end of which the notation $\frac{\Delta L}{\Delta P}$ is placed represents the elasticity of the muscle in the successive cycles. The curve at the end of which the notation $\frac{\Delta L}{\Delta \tau}$ is placed represents the plasticity of the muscle at the moment of the maximum charge.

It is evident from the comparison of the plasticity curves reproduced in figs. 11, 12, 16 and 21 that these curves resemble each other in their general features. The chief difference is in the scales upon which these tracings are drawn. A comparison of these scales proves that the plasticity increases when the rate of increase of the load grows larger. It is obvious that this result is inherent to the method I employed to measure the plasticity, and does not lie in the properties of the muscle. We conclude, therefore, *that the atonic muscle at the moment it is cut out of the body is a very plastic body, but that physical*

contrivances of different kinds, e.g., continued pull, cyclical variation of the load, vibration, &c., tend to eliminate the plasticity and to reduce the atonic muscle to a purely elastic body.

Finally, I will try to formulate in clinical terms the result of this part of my researches. The muscle, as soon as it becomes atonic, gradually increases in length. This increase is caused by the continued pull to which it is exposed by the tonus of its antagonists, by the stretching to which it is subjected during movement, by the vibrations to which it is subjected during changes in posture of the body, &c. The final length to which the muscle tends is probably identical with the

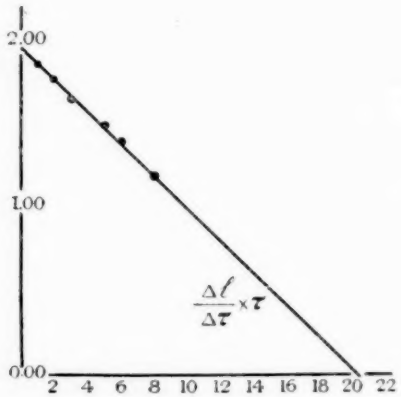

FIG. 20.—Graphic representation of the result of Experiment 16. The constant of plasticity during each interva succeeding a cyclical variation of the load. The numbers along the abscissa denote the successive cycles.

post-mortem length. In consequence of its increased length the muscle hangs loose and flabby between its insertions; in displacing the muscle, as a whole, to and fro across its length no active resistance is felt. Pressed between thumb and index the muscle feels solid, like a strand, has not the softness of the tonic muscle.

(*B*) *The Temperature Variable.*

The influence of the temperature upon the length of the atonic muscle has often been studied, and after the classic experiments of Brodie and Richardson [14] and the more recent work of Jensen [25] and others, my own researches are only of subordinate interest. I used

for these experiments the M. gastrocnemius of the frog. The muscle was cut out of the body and suspended in a small moist chamber. The moist chamber was surrounded by a closed mantle, in which two streams, one of cold water and one of hot water, could circulate. In this way the temperature of the moist chamber was varied. A thermometer was placed in the moist chamber, quite near the muscle, so that the fluid which kept the muscle wet formed a thin continued film round the muscle and the thermometer. The muscle was fastened to a length recorder, which registered the length of the muscle upon a rotating drum, upon which the time was also recorded. Every minute I noted the temperature of the thermometer in contact with the muscle. The curves recorded in this way are by intermediation of the time length-

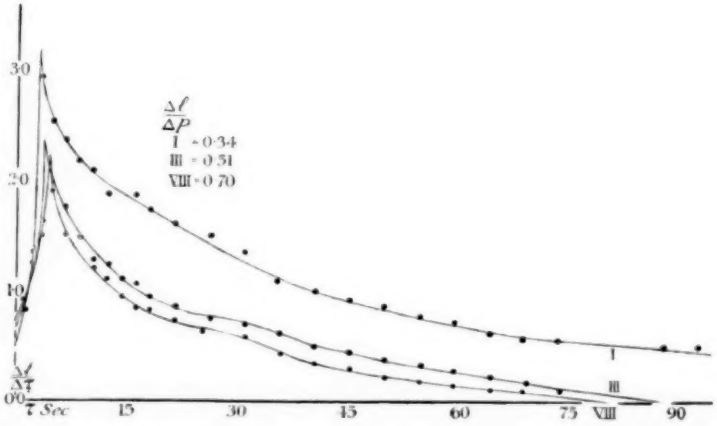

FIG. 21.—Graphic representation of the results of Experiment 16. The plasticity as a function of the time during the loading of the muscle—first, third and eighth extension curve.

temperature diagrams. This method is rather rough, and the construction of the thermostat too simple to give very exact results, but for my purpose it was sufficient.

The units adopted in this part of the researches are: 1° C. as the unit of temperature [T]; one minute as the unit of time [τ] and 1 mm. as the unit of length [L, l]. The enlargement by the length recorder amounted to 5·55, so that the real unit of length is $\frac{1}{5 \cdot 55}$ mm. The increase in length of the muscle is considered as a positive variation of L [+], the decrease of the length as a negative one [−].

The aim of these experiments was to establish a condition of uniformly varying temperature. The tables show that the condition of uniformity is not fulfilled; the curves, however, which I obtained are notwithstanding fairly regular. This result is only caused by the fact that the influence of the rate of variation of the temperature is not very marked upon the form of the length-temperature diagram. On the other hand, there is a compensation factor, viz., that the muscle, as a whole, follows but slowly the variation of the temperature of the ambient medium.

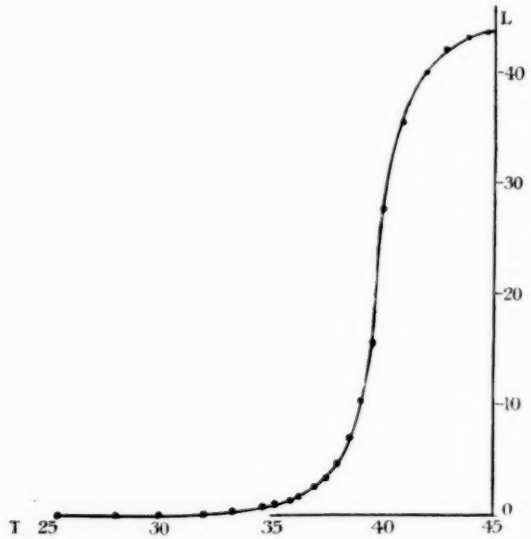

FIG. 22.—Length-temperature diagram of Experiment 1.

The muscle shortens in consequence of the increase of the temperature, until a first maximum is reached. This maximum lies in my experiments in the neighbourhood of 45° C. Beyond this first maximum I have not extended my experiments.

Experiment 1, *December* 18, 1906.—The frog was killed at 9.45 a.m.; at 10 a.m. the muscle was cut out, suspended in the moist chamber and charged with 14·9 grm. The experiment began at 10.17 a.m., but previously I performed ten cyclical variations with the load, partly to free the muscle from the after-phenomenon. It follows from Table XXII, which contains the result of the experiment, that by these cyclical

variations of the load the muscle is brought to a state of rest. More strictly speaking I ought to say *that the muscle has not come to a state of rest, but oscillates between very narrow and fixed limits.* The third row of Table XXII contains under $\left[\frac{\Delta T}{\Delta \tau}\right]$ the rate of the variations of the temperature, i.e., the increase or decrease of the temperature per minute. The mean value of $\left[\frac{\Delta T}{\Delta \tau}\right]$ during the interval 27° C. to 45° C. is about 0·3° C. per minute. The thermic shortening of the

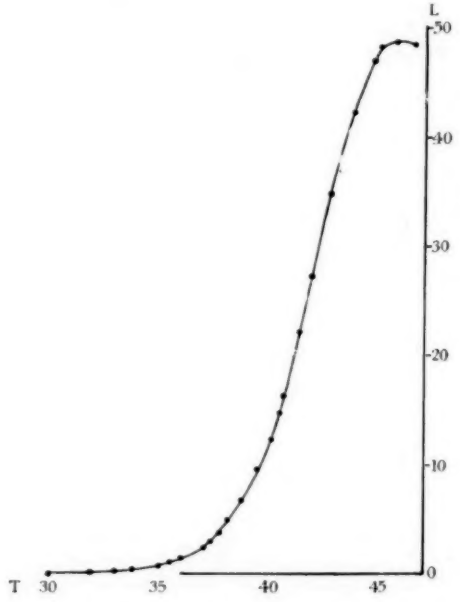

FIG. 23.—Length-temperature diagram of Experiment 2.

muscle begins between 25·4° C. and 27° C., and the first maximum is reached at 44·7° C.; at that moment the total shortening is about 21·5 per cent. of the initial length. Fig. 22 is the length-temperature diagram. This curve is fairly regular and **S**-shaped, a result already found by the earlier observers.

Experiment 2 is of the same kind. The mean value of $\left[\frac{\Delta T}{\Delta \tau}\right]$ during the interval of 30° C. to 45° C. is about 0·6° C., double the rate of increase of the temperature in the first experiment. Table XXIII contains the measurements of the curve, and fig. 23 is the length-

temperature diagram. In this case the curve is more flat and the S shape not quite so pronounced as in fig. 22. This result leads to the supposition that the rate of increase of the temperature might influence the form of the length-temperature diagram.

I tried to verify this supposition in the following way: First I took the left M. gastrocnemius of the frog and warmed the muscle rather slowly, then I took the right gastrocnemius and raised the temperature

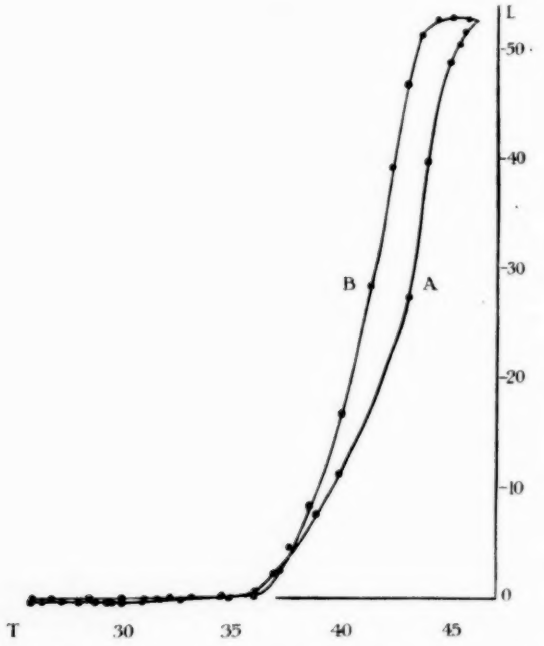

FIG. 24.—Curve A, length-temperature diagram of Experiment 3. Curve B, length-temperature diagram of Experiment 4.

more quickly. Tables XXIV and XXV contain the result of experiments 3 and 4, and fig. 24, curves A and B, are the pertaining length-temperature diagrams.

Experiments 5 and 6 are of the same kind. The result of the experiment is represented by Tables XXVI and XXVII, and fig 25 reproduces the length-temperature diagrams. The difference between the A and B curves is of the same nature as the difference between the

curves of the Experiments 1 and 2. We conclude, therefore, *that the rate of increase of the temperature influences the form of the length-temperature diagram.* One might object to this experiment on the ground that the two gastrocnemii of the same frog, though originally equal, are no longer alike at the moment of the experiment, because the experiment with the second gastrocnemius always began one to two hours after the experiment with the first muscle. The experiment

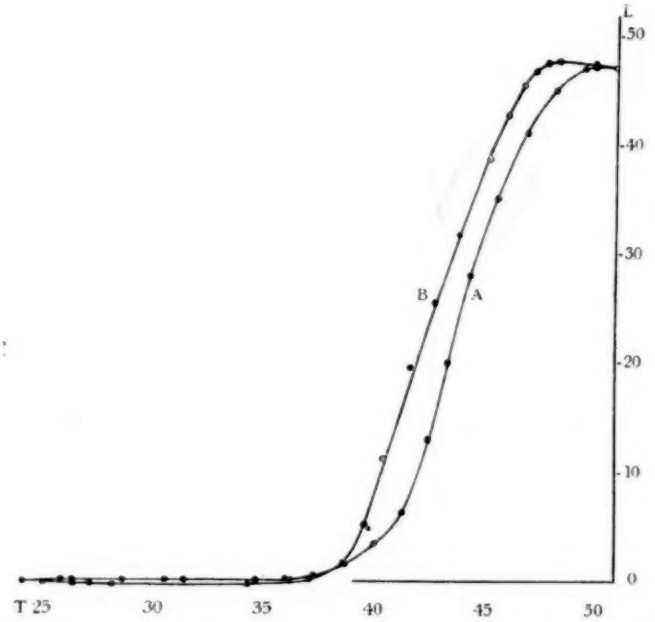

FIG. 25.—Curve A, length-temperature diagram of Experiment 5. Curve B, length-temperature diagram of Experiment 6.

shows, however, that in both cases the same final length was attained. This fact is in favour of the view that the difference between the A and B curves is chiefly caused by the difference in the rate of increase of the temperature.

The result at which we arrived may also be formulated in a somewhat different manner. We may conclude *that only the final state is fixed by the temperature of the muscle, but that the length of the muscle, at a moment intermediate between the beginning and the end of the shortening, is not merely determined by the temperature, but also by*

the way in which this temperature is reached. Formulated in this manner, we can prove the results of the foregoing experiments by cyclical variations of the temperature.

Experiment 7, *December* 1, 1906, is composed of three non-closed thermal cycles. The measurements of the original tracing are combined in Table XXVIII, and the length-temperature diagram is reproduced in fig. 26. First the temperature is raised from 12·4° C. to 32·5° C., with a mean increment of 0·48° C. per minute. Then the temperature falls from 32·5° C. to 21·5° C., with a mean rate of decrease of 1·37° C. The rise

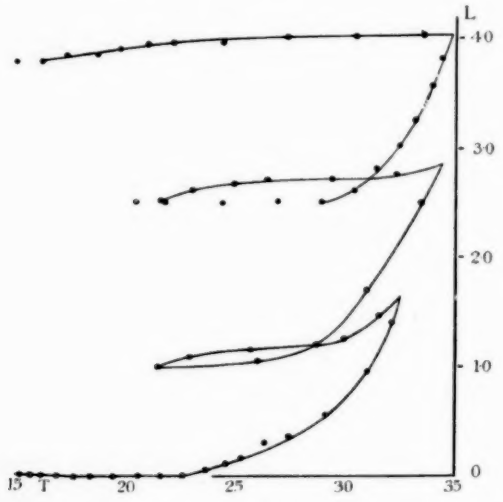

FIG. 26.—Length-temperature diagram of Experiment 7.

of the temperature is accompanied by a shortening of the muscle [rising curve], the fall of the temperature is followed by a lengthening of the muscle [falling curve]. In the first cycle the falling curve lies above the rising curve; this result is a general one, the falling curve always lying above the preceding rising curve, and we conclude, therefore, *that the increase of the temperature produces a shortening of the muscle, and that this shortening is partly permanent.*

The curve corresponding to the falling temperature is also **S**-shaped, but as the temperature at which the reversion from rising to falling temperature takes place becomes higher, the falling curve loses more and more its **S**-shaped form; at last the falling curve is nearly a horizontal line. This fact is illustrated by Experiment 8, where the

points of the reversion of the temperature lie higher than in Experiment 7. We conclude, therefore, *that the rise of the temperature produces a gradually increasing deformation of the muscle.*

The second thermal cycle of Experiment 7 is composed by an interval of increasing temperature from 21·5° C. to 33·5° C., with a mean increment of 1·38° C. per minute. This interval of rising temperature is followed by an interval of decreasing temperature from

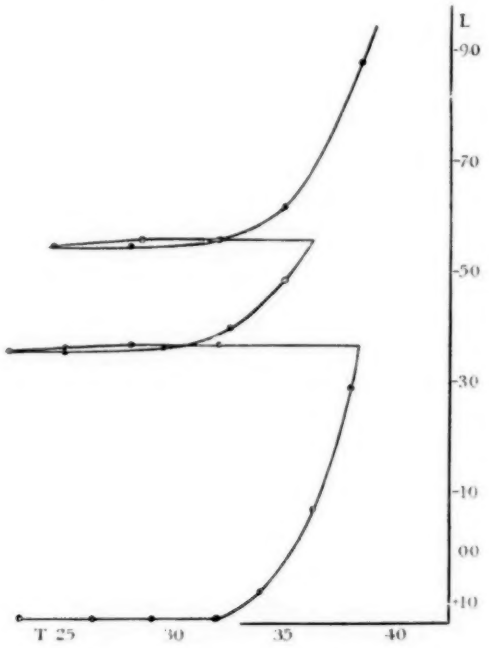

FIG. 27.—Length-temperature diagram of Experiment 8.

33·5° C. to 20° C., with a mean decrement of 1·86° C. The rising curve of the second cycle cuts the falling curve of the preceding cycle and forms a loop.

The third thermal cycle begins with an interval of increasing temperature, from 20·5° C. to 34·4° C., followed by an interval of decreasing temperature, from 34·4° C. to 15° C. The mean value of the variation of the temperature during the rising interval is 1·40° C., during the falling interval, 1·76° C. per minute. The second loop forms

a peculiarity. This loop is incomplete. As soon as a temperature of 21·7° C. is reached, the muscle ceases to lengthen, notwithstanding the temperature falling further to 20·5° C. At that moment the third thermal cycle begins, and the temperature again increases. The muscle, however, remains at constant length until the temperature reaches 29° C.; then a new period of shortening sets in. A part of the second loop, therefore, cannot be realized, in consequence of the permanent thermal deformation of the muscle.

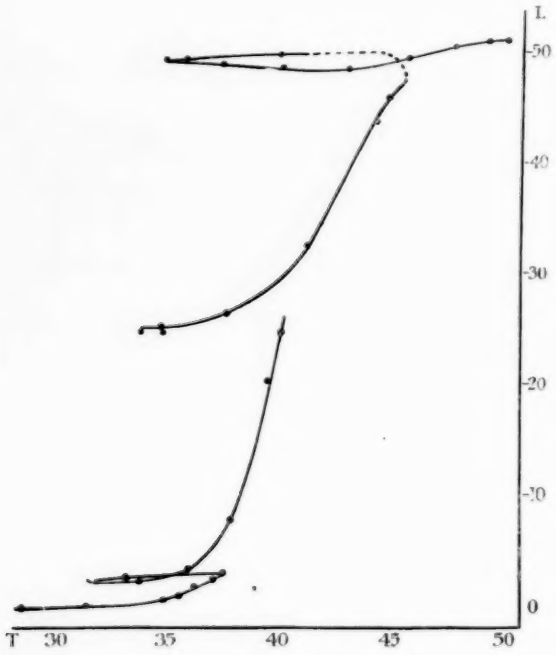

Fig. 28.—Length-temperature diagram of Experiment 9.

Experiment 8 is of the same nature as Experiment 7. The points of reversion from rising to falling temperature, however, lie higher. Table XXIX contains the measurements, and fig. 27 is the reproduction of the length-temperature diagram of this experiment.

The last experiment of this kind is No. 9. It is composed by three thermal cycles. The second loop could not be realized, and the third loop has a somewhat anomalous appearance. This is caused by the

well-known fact that in the neighbourhood of the first maximum, increasing temperature may be accompanied by a lengthening of the muscle. Table XXX gives the measurements, fig. 28 is the pertaining length-temperature diagram, and fig. 29 the original tracing. We conclude from these experiments *that the length of the atonic muscle is not entirely determined by its temperature, and inversely that a fixed temperature does not agree with a determined length of the muscle.* In other words, the length of the muscle is greatly determined by its physical and chemical deformation, and the amount of this deformation at a certain moment depends not only upon the actual temperature of the muscle, but also upon the way by which this temperature is reached.

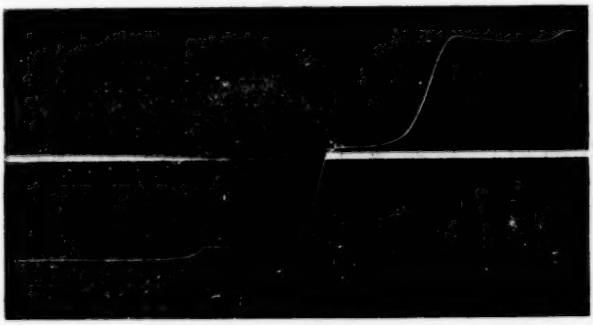

FIG. 29.—Reproduction of the myograph of Experiment 9. The enlargement is about 3·7 times.

Finally, I combined in a table the quantities, which are of interest for the knowledge of the process of thermal shortening. Ten of my experiments were suitable for this purpose. For each of these quantities I determined the arithmetical mean of these ten observations. In this way I found that the thermal shortening of the frog's gastrocnemius[1] begins between 28·5° C. and 29·5° C., that the first maximum of the shortening is reached in the neighbourhood of 46° C. and that the total amount of the shortening at that moment attains to 26 per cent.

The question of the thermal coefficient of the muscle during the interval preceding the thermal shortening is much discussed. If we define the term "thermal coefficient" as the variation in length in millimetres for 1° C. per centimetre length of the muscle I find :—

[1] I used for these experiments *Rana esculenta*, and the experiments were performed in winter.

$\frac{\Delta L}{\Delta T} \times \frac{1}{L} < 74 \times 10^{-5}$ for the interval 12° C. to 27° C.

[Experiment 1, dated December 18, 1906.]

" $< 60 \times 10^{-5}$ for the interval 12·4° C. to 22·7° C.

[Experiment 7, dated December 1, 1906.]

" $< 84 \times 10^{-5}$ for the interval 9·8° C.—25·3° C.

[Experiment 10, dated December 12, 1906.]

These observations prove that the thermal coefficient of the muscle is extremely small. In some experiments I found the phenomenon described by Brodie and Richardson, viz., that the muscle during the interval of temperature below the temperature at which the thermal deformation is manifest, may lengthen in consequence of a rise in temperature. I observed this phenomenon in some cases in the neighbourhood of 20° C. In these cases, however, it was the after-lengthening, which was again activated by the rise of the temperature, for I never saw the phenomenon when I previously eliminated the after-phenomenon by cyclical variations of the load. Moreover, the magnitude of the lengthening agreed with the lengthening caused by the after-phenomenon and was by no means of the order of magnitude of the thermal coefficient. We conclude, therefore, *that the thermal coefficient of the atonic muscle is extremely small within the limits of temperature which are of physiological interest, or small variations of the temperature round the physiological norm do not sensibly influence the length of the atonic muscle.*

Chapter II.—The Tonic Muscle.

My researches on the atonic muscle have proved that the muscle possesses two fundamental properties, viz., elasticity and plasticity. The aim of this second part of my investigation is to trace the rôle which these two properties play in the function of the tonic muscle. To that purpose, I make use of my experimental material of the years 1899, 1900 and 1901. A part of this material was published in *Archiv für Physiologie*, Jahrgang. 1901 and 1902, but the analysis given there was incomplete. It was the study of the atonic muscle which enabled me to analyse more completely the function of the tonic muscle. The morphological researches of the latter years also contributed to this result, since it was demonstrated by Boeke that striped muscle possesses a double motor innervation. He showed that the striped

muscle is not only innervated by the well-known axone of the motor cell of the anterior horn, but also by a motor sympathetic fibre. This fact makes the image of the simple proprioceptive reflex arc more complicated than it was before. For the sake of brevity and clearness I propose to give first a scheme (fig. 30) of the proprioceptive reflex arc, in which are incorporated the joint results of the morphological researches of Boeke [2], [3], [4], [5], [6] and of my own physiological investigations.

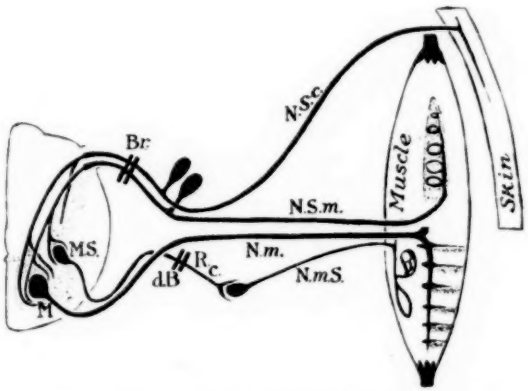

FIG. 30.—Scheme of the proprioceptive reflex arc: Br., place at which Brondgeest interrupted the proprioceptive reflex arc; d.B, place where de Boer interrupted the proprioceptive reflex arc; M, motor cell of the anterior horn; M.S., sympathetic motor cell; N.*m.*, efferent nerve, ending along the edges of the anisotropic discs; N.*m*.S., sympathetic efferent nerve; N.S.c., afferent nerve from the skin; N.S.*m.*, afferent nerve from the muscle; the terminal of the nerve in the muscle forms a muscle spindle; R.c., grey ramus communicans.

The receptors of the proprioceptive reflex arc are situated in the muscle, in the tendon, at the transition of the muscle into the tendon, in the fasciæ, the joints and the periosteum. The adequate stimuli for these proprioceptors are extension of the muscle and the tendon, stretching of the fasciæ, movements in the joints, pressure upon the periosteum, &c. The receptors are connected with afferent fibres which enter the cord along the posterior roots. Here the afferent fibre splits up into collaterals, which terminate around two different and spatially separated motor cells. One of them is the well-known large motor cell of the anterior horn. The axone of this cell is usually considered to

terminate in the motor end-plate of the muscle-fibre, but according to the researches of Boeke does not really end there. Fine filaments leave the net of neurofibrils situated in the end-plate and run parallel to the striped muscle-fibre. They in that way give off finest filaments at a right angle, which extend along the edge of the anisotropic disc, where they seem to end. It appears, therefore, that a very close connexion exists between the axone of the motor cell of the anterior horn and the striped apparatus of the muscle.

The other of the two motor cells is a sympathetic cell. The axone of this cell leaves the cord along the anterior root and passes over into the sympathetic chain by way of the grey ramus communicans. When this sympathetic fibre reaches the muscle, it forms a non-myelinated sympathetic plexus, from which fine filaments enter the muscle-fibre. These filaments end in fine nets of neurofibrils, which are embedded in a small sarcoplasmatic end-plate. In general the sympathetic cell does not lie in the same level as the motor cell of the anterior horn. It seems from experiments of de Boer [7], [8], [9], and of Keere, Hiramatsu, and Naito [26] that the sympathetic cells are situated in the thoracic part of the cord, and that they belong to the thoracic sympathetic system.

The morphological researches of Boeke lead, therefore, to the conception of *the duality of the striped muscle, viz., a sarcoplasmatic mass, innervated by a sympathetic fibre analogous to a smooth muscle, in which is embedded a striped apparatus. This apparatus is in close connexion with or forms the termination of the axone of the motor cell of the anterior horn.* The notion of the duality of the striped muscle was already held several years ago by Fano [18], Botazzi [11], [12], Zoethout [49], and others, and lastly again by Pekelharing [35], [36] and by de Boer [9].

The question whether the exteroceptors originating in the skin are also connected with the sympathetic motor cell in the cord is not settled by my experiments. I am inclined to believe that of all the different kinds of exteroceptors only the thermoceptors of the skin may have a direct connexion with the sympathetic motor cell, and that all the other exteroceptors act only indirectly upon the sympathetic motor cell by the intermediation of an intercalated proprioceptive reflex arc.

The experiments were made with the M. gastrocnemius of the frog and the cat. First I will describe the experiments with the frog's gastrocnemius. The tendon of the muscle was cut through and the muscle itself slightly loosened from the surrounding tissue. The nerves and

blood-vessels were spared, so that the muscle remained in quite normal relation with its proprioceptive reflex arc. A light recipient was fixed to the tendon by means of a hook. A stream of mercury flowed in the recipient with constant velocity, so that the charge of the muscle increased uniformly with the time. As soon as the charge reached a certain maximum the inflow was stopped, and then after some seconds the muscle was unloaded abruptly. This experiment was repeated several times with approximately equal intervals. The experimental condition established in that way is therefore one of successive cyclical variations of the load. The variation of the length of the muscle under the influence of the varying load was recorded upon a rotating drum upon which the time was also noted. The curves obtained in this manner are extension curves of the tonic muscle.[1]

The experiment of making extension curves of the tonic muscle by means of a uniformly increasing weight is not as simple as it seems to be, for in the first place the weight acts as a purely physical factor, as a load that extends the muscle, but in the second place it acts as a physiological agent. The stretching of the muscle by means of the increasing weight forms an adequate stimulus for the proprioceptors of the muscle to which it answers with a slow contraction. *The extension curve of the tonic muscle is therefore an extension curve, not of a resting muscle but of a contracting one.*

The fact that the tonic muscle, when stretched by an increasing weight, really contracts is demonstrated by the experiments of Buytendijk [15] and Samkow [39]. Buytendijk observed the action current of the tonic M. vastocrureus of the cat, and found that the intensity of this current augments, when the load attached on the tendon increases, till a certain maximum is attained.

The units used in this part of the research are: 1 second as the unit of time [τ]; 1 cm. as the unit of length [L, l]; 1 grm. as the unit of weight [P, p].

The analysis of the extension curve of the tonic muscle is based upon the following considerations. The extension curve is by intermediation of the time a length-load diagram, i.e., the graphic record of the relation between the length of the tonic muscle and the load attached on its tendon. It is therefore possible to determine directly from those tracings the increase in length of the muscle per gramme increase of the load. This quantity I have defined, in my work

[1] A full account of the technique is given in *Engelmann's Archiv*, Jahrgang 1901, S. 106.

published in *Engelmann's Archiv*, as the *tonus* of the muscle, in accordance with the earlier observers. The study of the atonic muscle has proved, however, that this quantity comprises two different elements, viz., an elastic elongation and an after-elongation, or, in other words, *that the tonus is the sum of an elasticity and of a plasticity.* The quantity defined as tonus has the advantage of being a directly measurable quantity, but the disadvantage that it does not allow of a more complete analysis of the properties of the tonic muscle. For the moment I will represent the tonus of the muscle at rest by the letter "*a*."

The stretching of the tonic muscle by means of a uniformly increasing weight stimulates the proprioceptors and causes the muscle to contract. This contraction produces a variation of the length of the muscle, the value of which per unit increment of the load may be represented by $\left[\frac{\Delta l}{\Delta P}\right]$. Hence the tonus of the muscle under the conditions of the experiment ought to be represented by $\left[a - \frac{\Delta l}{\Delta P}\right]$. We have therefore $\left[a - \frac{\Delta l}{\Delta P}\right]$ = muscular tonus = the increase in length of the tonic muscle per gramme increase of the load. My earlier observations have proved that there exists a connexion between the stretching weight and the tonus, viz., *when the weight increases in geometrical progression the tonus decreases in arithmetical progression.*

Expressed in mathematical symbols we find—

$$e^{C_2\left(a - \frac{\Delta l}{\Delta P}\right)} = C_1 P \text{.....IA ; or,}$$

$$C_2\left(a - \frac{\Delta l}{\Delta P}\right) = \text{lgn. } C_1 P \text{ ...IB.}$$

in which formulæ—

$\left(a - \frac{\Delta l}{\Delta P}\right)$ = tonus,
P = the stretching weight,
C_1 and C_2 = two constants.

The stretching weight P functionates as a stimulus for the proprioceptors, so that we may also say that $[C_1 P]$ represents the value of the stimulus, and $\left[C_2\left(a - \frac{\Delta l}{\Delta P}\right)\right]$ the corresponding value of the tonus. Formula I states, therefore, *that there exists a logarithmic relation between the stimulus and its effect.*

The physiological interpretation of the result of my experiment makes it necessary to introduce the threshold value Δ into the formula, whereby this formula assumes the form :—

$$C_2\left(a - \frac{\Delta l}{\Delta P}\right) = \text{lgn. } C_1(P - \Delta) \text{.....IC,}$$

$[P-\Delta]$ being the excess of the stimulus above the threshold value. Formula IC may be integrated, if we substitute an integration for a summation. We find as the result of the integration—

$$l = a\,P - \frac{1}{C_1 C_2}[C_1(P-\Delta) + \lg.\ C_1(P-\Delta) - 1\ \}]\ \text{II};$$

this formula expresses the relation between the increase in length of the tonic muscle [l] and the increase of the weight [P].

Formula II states more particularly that the increase in length of the tonic muscle may be considered as the difference of two quantities, viz.—

$$[a\,P] \text{ and } \frac{1}{C_1 C_2}[C_1(P-\Delta)\ \{\ \lg.\ C_1(P-\Delta) - 1\ \}].$$

The first of these quantities, according to the definition given of a, is the total increment in length of the muscle caused by the increase of the weight P, if the muscle did not contract. The second quantity, in agreement with the genesis of the integrated formula, represents the shortening of the muscle in consequence of the slow contraction, caused by the stimulation of its proprioceptors. The increase in length of the tonic muscle under the conditions of the experiment is therefore the sum of a purely physical elongation, and a shortening caused by a physiological process.

This result conforms with the experimental data of the first part of this research, for if the proprioceptive reflex arc is broken, the physiological effect vanishes, and formula II is reduced to $l = a\,P$, i.e., the increment in length of the atonic muscle is the sum of the elastic elongation and of the elastic after-elongation. For the case the after-phenomenon is eliminated, $l = a\,P$ represents the law of Hooke. In the third part of this investigation I will show that the formula

$$\frac{1}{C_1 C_2}[C_1(P-\Delta) + \lg.\ C_1(P-\Delta) - 1\ \}]$$

may represent the relation between the stimulus and the lift of a muscle contracting under isotonic conditions.

First I will prove that the experimental results may be represented by the analytical expression IC. The experiments used to that purpose were for the greater part already published in *Engelmann's Archiv*, 1901. The numbers placed in brackets behind the numbers of the experiments refer, therefore, to the Tables and to the reproductions of the original tracings in that publication. I repeated, however, the measurement of the length-load diagrams and corrected thereby little faults in my earlier measurements. I moreover extended my measurements to parts of the curve which are now of interest for the

more complete knowledge of the problem, but which at that time were neglected.

Experiment 1 [*Table II*], *September* 15, 1899. — The frog used for this experiment was intact, but by wrapping up the whole animal in wet cotton-wool unintended stimuli were fairly well eliminated. The successive extension curves of the tonic M. gastrocnemius were made with approximately equal intervals of some minutes. The curves thus obtained resemble each other closely, and the course of the whole experiment was not troubled in any way. In some other experiments the cord was previously cut through, but, at least in the frog, there was no appreciable difference in the tracings in either case, except a slight depression of the tonus by shock, shortly after the section of the cord. Table XXXI gives the measurements of the fifth extension curve. The first row of this table contains the time [τ], the second row the charge [P], the third row the increment in length [L] of the tonic muscle, caused by the weight [P] at the moment [τ]. In the fourth row I give the tonus of the muscle, i.e., the directly measured increase in length of the muscle per gramme increase of the load at the moment [τ]. Hence *the tonus is the directly accessible quantity, the value of which is not influenced by any aprioristic considerations.* The fifth row contains the quantity $C_2 \left(a - \frac{\Delta L}{\Delta P}\right)$, and the sixth row the quantity lgn. $C_1 (P - \triangle)$. According to equation IC, these two quantities ought to be equal. The seventh row contains the difference between the quantities of rows 5 and 6.

$\triangle$ is the threshold value of the stimulus, which is always determined by means of a simple graphic extrapolation. The constants C_1 and C_2 are determined in such a way that equal worth is attributed to each observation upon the value of these constants.

Experiment 2 [*Table III, fig.* 1], *December* 6, 1899.—The conditions of this experiment are the same as of Experiment 1. Table XXXII contains the measurements of the ninth extension curve.

Experiment 3 [*Table VIII, fig.* 2], *November* 11, 1899.—In this case the cord was cut through between the first and second vertebræ. Table XXXIII contains the measurements of the fourteenth extension curve about one hour after the transection of the spinal cord. We conclude from these experiments *that within the limits of experimental error formula IC represents the relation between the increase of the weight and the decrease of the tonus.*

This relation being fixed, I calculated "a" with the aid of the inte-

grated formula II, using the constants $\triangle$, C_1 and C_2 of Tables XXXI, XXXII and XXXIII.

TABLE A		TABLE B		TABLE C	
τ	a	τ	a	τ	a
Sec.		Sec.		Sec.	
3·0	20×10^{-3}	3·0	$17{\cdot}8 \times 10^{-3}$	3·0	$20{\cdot}4 \times 10^{-3}$
6·2	21·2	6·2	20·7	6·2	20·4
12·6	19·7	12·6	20·4	12·6	19·1
25·4	17·7	25·4	18·6	25·4	16·7
33·5	16·8	35·0	17·6	30·9	16·0

We conclude, therefore, *that "a" is not constant.* The deviation of a from constancy induced me to investigate the beginning of the extension curve, especially the interval 0 sec. to 3 sec.

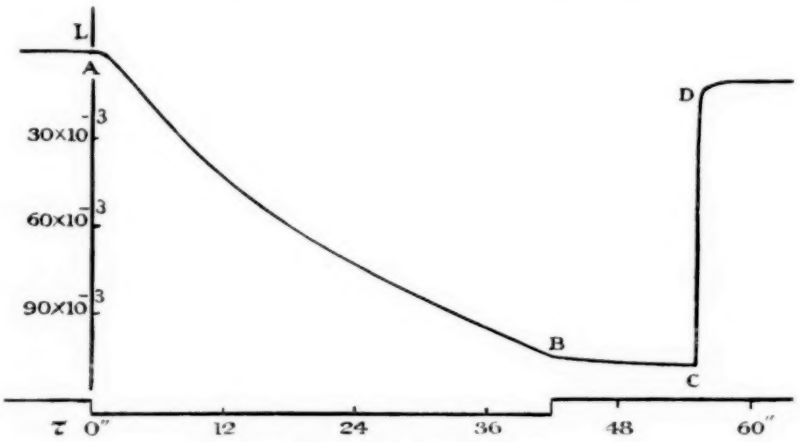

FIG. 31.—Length-load diagram of the tonic muscle.

Experiment 4 [*Table I*], *November* 10, 1899.—The frog used for this experiment was intact and wrapped up in cotton-wool. Table XXXIV contains the measurements of the thirteenth extension curve, and fig. 31 is the graph of the length-load diagram. Along the abscissa the time is noted in seconds, and along the ordinate the increment in length of the muscle in centimetres. Because the weight increases uniformly with the time, the curve ABCD is a length-load diagram. At A the inflow of the mercury in the recipient begins and the muscle lengthens under the

influence of the increasing weight. At B the inflow is stopped, and the muscle from that moment onwards under the influence of a constant load, slowly continues to lengthen. At C the muscle is abruptly unloaded and shortens, at first quickly till D, and then slowly creeps back. After a short interval of rest the cycle is repeated.

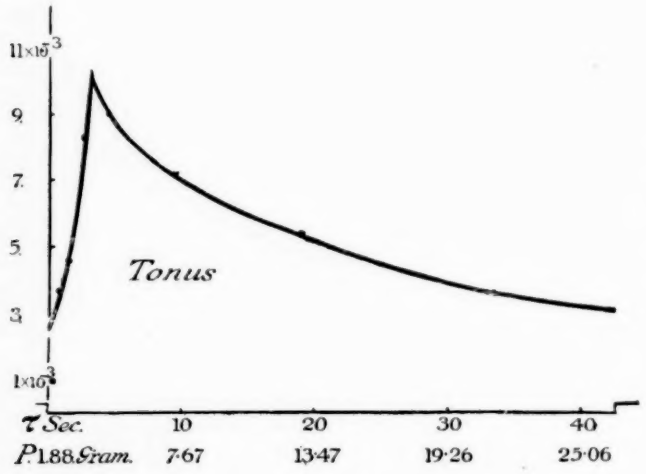

FIG. 32.—Tonus diagram of Experiment 4.

Fig. 32 is the tonus diagram. It appears from this graph that the tonus at first quickly increases and then decreases. The phase of increasing tonus lasts in this case about 3 sec., and the phase of decreasing tonus 40 sec. The initial and rapid increase of the tonus under the influence of the increasing weight is a fact found in all the following experiments. We conclude, therefore, *that under the influence of a uniformly increasing weight the tonus at first quickly increases and then slowly decreases.* For the interval of decreasing tonus the relation IC holds good, as may be seen from Table XXXV.

If we calculate a by means of formula II we find that a at first increases and then decreases. According to our definition of a this quantity is the sum of an elasticity and of a plasticity, and the question now arises which of these two quantities varies. There are two moments at which the elasticity of the muscle may be determined directly from the length-load diagram. Firstly, the elasticity can be determined in the neighbourhood of A (fig. 31), and I choose for that

purpose the interval 0·5 sec. to 1 sec. The interval 0 sec. to 0·5 sec. is in most cases not suitable for the determination of the elasticity, because passive resistances, residing in the apparatus and probably also in the muscle, oppose themselves to the extension of the muscle. The elasticity, as determined from that interval, is therefore evidently too small. At the interval 0·5 sec. to 1 sec. the passive resistances no more make their influence felt, and the charge is still small, so that the after-phenomenon vanishes against the elastic elongation. In the second place we may determine the elasticity from the part CD (fig. 31), because CD is the shortening caused by a known and abrupt variation of the charge. The position of D, however, where the direct elastic shortening goes over into the after-phenomenon, is not quite fixed, but this uncertainty is largely counterbalanced by the advantage that the simultaneous variation of the length and the load are fairly considerable. At the moment the experiments were made, I could not foresee that this part of the tracing would be of interest, so that a certain number of my tracings are not suitable for the determination of the elasticity from the part CD. I found, however, in my material a sufficient number of tracings which allowed of the determination of the elasticity also from that part of the curve. The result of the determination of the elasticity from both parts of the tracing is that the elasticity, as determined from the beginning and from the end of the tracing is the same in both instances. We conclude, therefore, *that the elasticity is constant and that the variation of a is due to a variation of the plasticity of the muscle.* This fact agrees with the results obtained in the first section of this research. There I found that the elasticity of the atonic muscle plays only a subordinate rôle, inasmuch as the elasticity varies but little and the problem is principally one of plasticity. It seems also in harmony with the results of modern physiology, which considers muscle-contraction chiefly as a process of deformation.

The plasticity of the tonic muscle is now determined in the following manner. First the constants Δ, C_1 and C_2 are fixed by means of formula IC. Secondly, $[a\ P]$ is calculated by introducing the values of these constants in formula II. Next, the elasticity is determined from the beginning and the end of the length-load diagram. If we keep in view that a is the sum of an elasticity and of a plasticity, we may determine the plasticity by introducing the value of the elasticity in the expression $[a\ P]$.

Fig. 33 is the plasticity diagram. I noted in this diagram the time along the abscissa and the plasticity along the ordinate. The

diagram shows that the plasticity of the muscle as well as the tonus at first quickly increase and then slowly decrease. When the inflow of the mercury is stopped the stimulation of the proprioceptors ceases, and the plasticity falls suddenly.

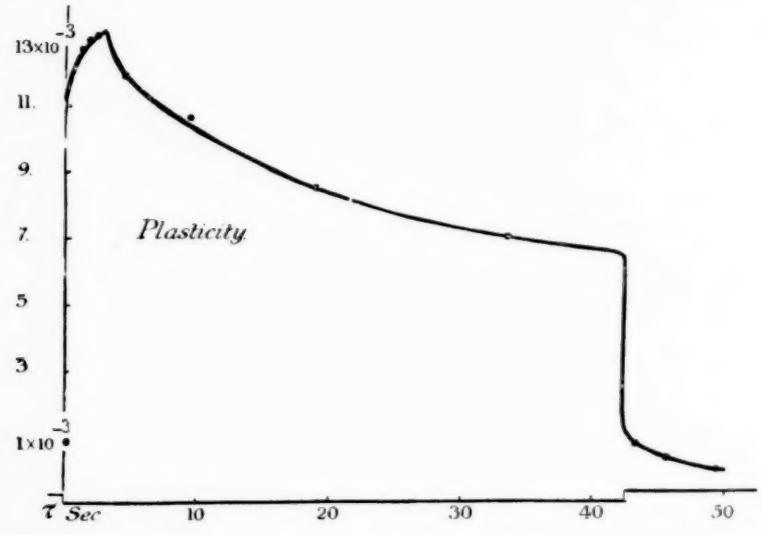

FIG. 33.—Plasticity diagram of Experiment 4.

Experiment 5 [*Tables IX, X*], *December* 2, 1899.—The frog used for this experiment was intact and wrapped up in cotton-wool. I recorded first a number of extension curves of the tonic muscle, and then I cut through the nervus tibialis. By the section of the nerve the proprioceptive reflex arc is broken, and the muscle becomes accordingly atonic. About half a minute after the section of the N. tibialis I began to record a set of extension curves of the atonic muscle. Table XXXVI contains the measurements of the last extension curve of the tonic muscle before the section of the nerve. The constants Δ, C_1 and C_2 were determined from the interval 3 sec. to 25 sec., and Table D shows how far the measurements agree with formula IC.

Fig. 34 represents the tonus diagram and figure 35 the plasticity diagram.

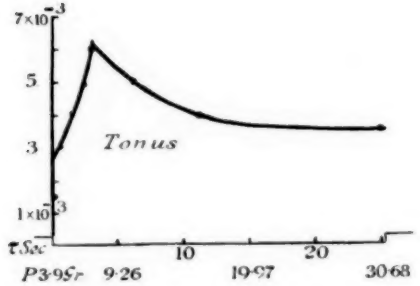

FIG. 34.—Tonus diagram of Experiment 5.

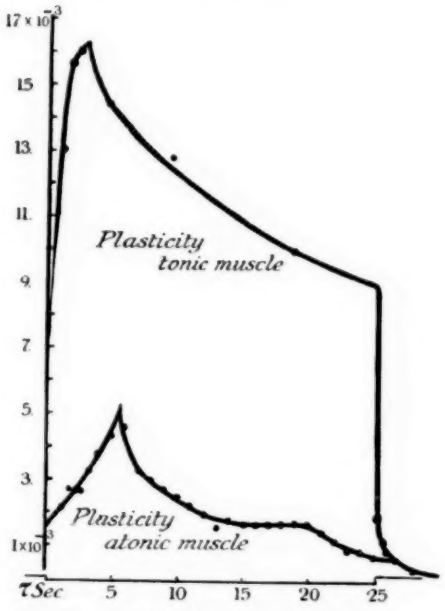

FIG. 35.—Plasticity diagram of Experiment 5.

TABLE D.

τ	$C_2\left(a-\frac{\Delta l}{\Delta P}\right)$	lgn. $C_1(P-\Delta)$	Diff.
Sec.			
3·0	− 0·720	− 0·717	+ 0·003
6·2	− 0·600	− 0·570	+ 0·030
12·6	− 0·480	− 0·481	− 0·001
25·0	− 0·420	− 0·429	− 0·009

$\Delta = 0{\cdot}3$ $C_1 = 7{\cdot}74 \times 10^{-3}$ $C_2 = -120$

The N. tibialis was cut through 45·2 sec. after the beginning of the last extension curve of the tonic muscle, the muscle being loaded at that moment with 30·9 grm. The interval immediately following the section of the nerve, from 45·2 sec. to 50 sec., is irregular, the course of the experiment being broken by contractions, but from 50 sec. to 65 sec. these contractions have ceased, and it is now possible to determine the course of the after-phenomenon. It appears from the comparison of the after-phenomenon before and after the section of the nerve that by the section of the nervus tibialis the after-lengthening is activated for a short while,[1] or in other words, that the interruption of the proprioceptive reflex arc exalts for a moment the plasticity of the muscle. The muscle is unloaded about 20 sec. after the section of the nerve. The experiment was so conducted that it was possible, without any doubt, to determine the shortening of the muscle caused by the abrupt diminution of the charge by 27 grm. The elasticity amounted in this case to $3{\cdot}09 \times 10^{-3}$, being the same as at the beginning, where it was $3{\cdot}04 \times 10^{-3}$. We see, therefore, that the section of the motor nerve affects immediately the plasticity of the muscle, but not the elasticity. The first extension curve of the atonic muscle is recorded about 25 sec. after the section of the nervus tibialis. In fig. 35 I reproduce the plasticity diagram derived from this extension curve, drawn on the same scale as the plasticity curve deduced from the immediately foregoing extension curve of the tonic muscle. It is evident from fig. 35 that by the interruption of the proprioceptive reflex arc, the plasticity of the muscle is considerably lowered.

Experiment 6 [*Table XI, fig. 3*], *December* 3, 1899.—This experiment is exactly of the same kind as No. 5.

Table XXXVII contains the measurements of the last extension curve of the tonic muscle and the effect evoked by the section of the nervus tibialis. Table XXXVIII reproduces the measurements of the first and second extension curves after the section of the nerve. The constants Δ, C_1 and C_2 are determined from the interval 3 sec. to 25·4 sec., and Table E shows the agreement with formula IC.

Figs. 36 and 37 are the reproductions respectively of the tonus and the plasticity diagrams.

The N. tibialis was divided 40·6 sec. after the beginning of the last extension curve of the tonic muscle, the muscle being loaded at that

[1] A. Benedicenti (*Arch. ital. d. Biol.*, 1897, vol. xxviii) also arrived at the result that the section of the nerve is followed by a permanent lengthening of the muscle.

moment with 28·09 grm. It was possible in this case to determine the course of the after-phenomenon during the interval following the section of the nerve. The measurements carried out upon that part of the curve falling within the interval 45 sec. to 56 sec. show that the section of the nerve strongly accentuates the process of after-lengthening. The quantity $\left[\frac{\Delta l}{\Delta \tau} \times \tau\right]$, if the moment at which the nerve is divided is taken as the zero of time, is, however, not constant, but at first increases and then decreases, probably striving towards constancy.

TABLE E.

τ	$C_2\left(\alpha - \frac{\Delta l}{\Delta P}\right)$	lgn. $C_1(P - \Delta)$	Diff.
sec.			
3·0	− 0·851	− 0·857	− 0·006
6·2	− 0·709	− 0·705	+ 0·004
12·6	− 0·568	− 0·565	+ 0·003
25·4	− 0·426	− 0·429	− 0·003

$\Delta = 0{\cdot}4$ $C_1 = 4{\cdot}24 \times 10^{-3}$ $C_2 = -129$

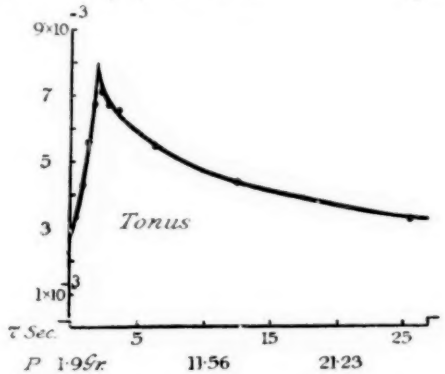

FIG. 36.—Tonus diagram of Experiment 6.

The muscle was unloaded about 27 sec. after the section of the nerve and the elasticity determined at that moment amounted to $3{\cdot}57 \times 10^{-3}$. The elasticity at the beginning of the last extension curve of the tonic muscle, i.e., before the section of the nerve, amounted to $3{\cdot}36 \times 10^{-3}$. We see, therefore, that the section of the motor nerve immediately alters the value of the plasticity of the muscle, but that the value of the elasticity remains at first constant.

The first extension curve of the atonic muscle was recorded about 72 sec. after the section of the nerve. The elasticity of the muscle determined from the beginning of this curve amounted to $2{\cdot}16 \times 10^{-3}$, and 84 sec. later, at the beginning of the second extension curve of the atonic muscle, the value of the elasticity is $2{\cdot}09 \times 10^{-3}$. The elasticity, therefore, shortly after the section of the motor nerve also

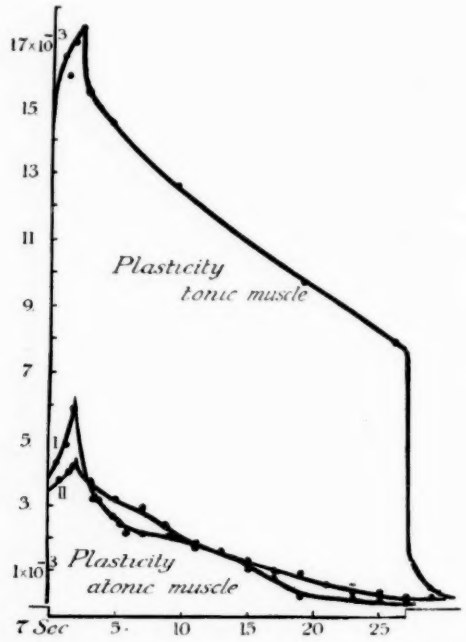

FIG. 37.—Plasticity diagrams of Experiment 6.

diminishes, but, as seen from Table XXXVIII, the diminution of the plasticity following the initial period of exaltation is the most striking effect of the interruption of the proprioceptive reflex arc.

Experiment 7, December 1, 1899.—This experiment is of the same kind as Nos. 5 and 6, but the section of the motor nerve took place after the muscle was unloaded.

Tables XXXIX and XL contain the measurements respectively of the last extension curve of the tonic muscle and of the first and second extension curves after the section of the N. tibialis. Figs. 38 and 39

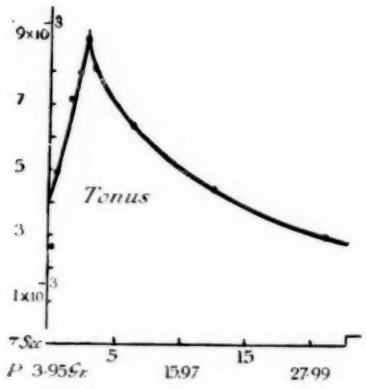

FIG. 38.—Tonus diagram of Experiment 7.

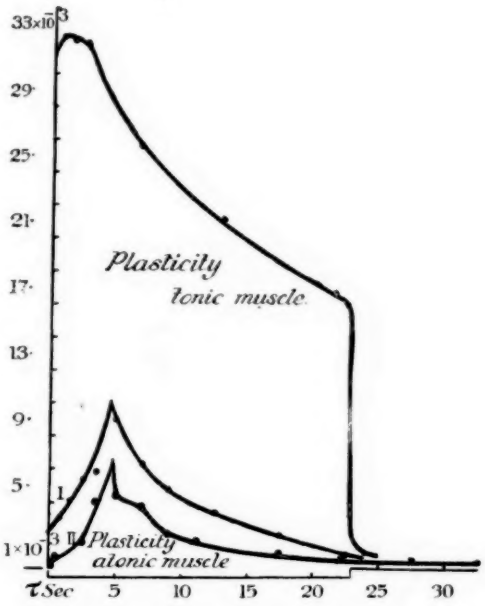

FIG. 39.—Plasticity diagrams of Experiment 7.

are the pertaining graphs. The constants Δ, C_1 and C_2 are determined from the interval 3 sec. to 22 sec., and Table F shows in how far this part of the curve agrees with the formula IC.

TABLE F.

τ	$C_2\left(a - \frac{\Delta l}{\Delta P}\right)$	lgn. $C_1(P - \Delta)$	Diff.
Sec.			
3·0	− 0·600	− 0·597	+ 0·003
6·2	− 0·458	− 0·452	+ 0·006
12·6	− 0·316	− 0·314	+ 0·002
22·0	− 0·213	− 0·217	− 0·004

$\Delta = 0{\cdot}3$ $\quad C_1 = 1{\cdot}27 \times 10^{-3}$ $\quad C_2 = -70{\cdot}5$

The elasticity, as determined from the beginning of the last extension curve of the tonic muscle, amounted to $2{\cdot}69 \times 10^{-3}$. The value found by the unloading of the muscle is $2{\cdot}66 \times 10^{-3}$, but this last determination was not quite certain.

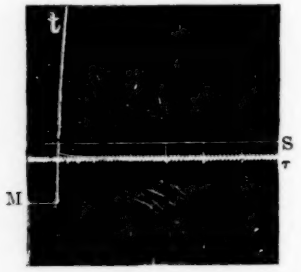

FIG. 40.—Myograph showing the after-lengthening of the M. gastrocnemius immediately after the section of the N. tibialis. M, the myograph: S, horizontal line drawn by the signal; t, the twitch evoked by the section of the nerve; τ, the time marked in tenths of seconds. After the contraction the line of the myograph runs through the time marking. By the reproduction this part of the myograph has become very indistinct, but in the original tracing the line was distinctly to be seen.

A short while after the unloading the muscle arrived at a state of rest, the length being constant; then the motor nerve was cut through, the muscle being loaded at that moment with only 3·95 grm. The section of the nerve is accompanied by a maximal contraction, after which the muscle does not return to its original length (fig. 40).

Immediately after the contraction the muscle begins to lengthen, and this lengthening exhibits all the characteristics of an after-phenomenon. It appears from the form of the after-lengthening curve that the muscle does not again reach its original length, but tends to a state of rest, at which its length is less than it was before the section of the nerve.

This experiment elucidates the much-discussed question, whether a tonic muscle after the section of its motor nerve becomes longer or shorter. It is evident that what happens after the section of the nerve depends chiefly upon the external conditions. For the section of the nerve acts as a supramaximal stimulus, which provokes a contraction, and meanwhile a permanent deformation (*vide infra*, Chapter III). When the load is heavy enough to eliminate this deformation and to reduce the muscle to its initial length, the muscle will become longer than it was before by the activation of the after-lengthening. When the load is not sufficient to neutralize the permanent deformation, the activation of the after-lengthening will in most cases not suffice to compensate the shortening caused by the permanent deformation. In that case the muscle will be shorter after the section of its motor nerve.

I determined in this case also the value of the quantity $\left[\frac{\Delta l}{\Delta \tau} \times \tau\right]$ and found that this quantity at first increases and then decreases in the same manner as in the preceding experiment.

The elasticity determined from the beginning of the first extension curve of the atonic muscle amounts to $2{\cdot}40 \times 10^{-3}$, and from the end of the curve at the moment of the unloading to $2{\cdot}80 \times 10^{-3}$; determined from the beginning of the second extension curve I found for the value of the elasticity $2{\cdot}40 \times 10^{-3}$, and from the end of the same curve, $2{\cdot}43 \times 10^{-3}$. There is also in this case shortly after the section of the motor nerve a slight diminution of the elasticity, but the fall of the plasticity is the prevailing result of the interruption of the proprioceptive reflex arc. We conclude, therefore, from these experiments *that the section of the motor nerve, by which the proprioceptive reflex arc is broken, exerts a twofold effect upon the striped muscle, viz., it immediately activates, though only for a few seconds, the after-lengthening; this rise of plasticity is followed by a considerable fall, which is permanent*, and may, under conditions described in the first part of this research, lead to the complete loss of the plasticity; *it leaves the elasticity at first unaltered, but within half a minute the elasticity also diminishes. The diminution of the elasticity, however, does not seem to be very considerable*, and, as, described in the first part

of this research, the elasticity may again slightly increase *pari passu* with the disappearance of the plasticity.

The next group of experiments was made with the M. triceps suræ of a cat, whose spinal cord was previously transected. Full-grown,

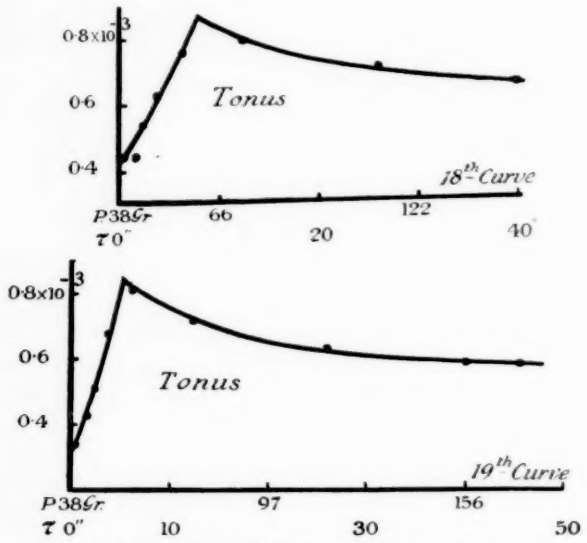

FIG. 41.—Tonus diagrams of Experiment 8.

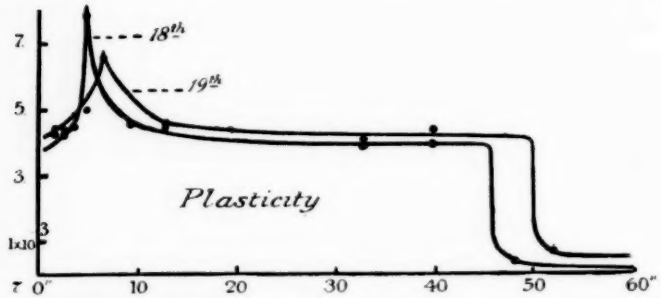

FIG. 42.—Plasticity diagrams of Experiment 8.

strong cats proved to be the most suitable for the experiments. The operation was performed under ether narcosis, but after the transection of the spinal cord no more ether was given. The transection of the cord was always followed by spinal shock, but after about one hour

the muscles partially regained their tonicity. The technique of these experiments is more difficult and more complicated than in the case of the frog, so that the experimental results are less exact. The methods however, were the same.[1]

The extension curve of the tonic M. triceps of the hind limb of the spinal cat exhibits exactly the same character as those of the frog, and the analysis of the tracings is based upon the same principle.

Experiment 8 [*Table XVIII*], *December* 4, 1900.—The spinal cord of the cat was transected at 11.30 a.m. between the last cervical and the first thoracic vertebræ. In the beginning of the experiment the extension curves are strictly linear, but as soon as the shock passes away the curves assume gradually the form characteristic for the tonic muscle. Tables XLI and XLII contain the measurements respectively of the eighteenth and nineteenth extension curves. The eighteenth is recorded at 1.23 p.m., and the nineteenth curve about three minutes later. Fig. 41 represents the tonus diagrams, and fig. 42 the plasticity diagrams.

Experiment 9 [*Table VII*], *December* 6, 1900, is of exactly the same kind. The transection of the spinal cord was performed between the sixth and seventh thoracic vertebræ at 10.30 a.m. The first extension curves are linear, but already the seventh curve shows a slight deviation from the straight line. Tables XLIII and XLIV contain the measurements of the fifteenth and sixteenth extension curves. The fifteenth curve was recorded at 1.48 p.m., and the sixteenth curve was registered about three minutes later. Figs. 42A and 43 are the tonus and the plasticity diagrams respectively.

We conclude from these experiments *that the statements made for the tonic muscle of the frog hold good also for the tonic muscle of the spinal cat*. There are, however, differences between the tonic muscle of the frog and that of the cat. The most striking difference resides in the form of the plasticity diagram. In the cat the increase of the plasticity takes place more abruptly, and also the subsequent fall is more sudden, until a level is reached, which remains nearly constant during the increment of the load. Moreover, in the cat the plasticity still more prevails over the elasticity than is the case in the frog.

The following group of experiments consists in making extension curves of the normal human triceps suræ with the myotonometer of

[1] A detailed description of the technique of these experiments is given in *Archiv. f. Physiol.* Jahrgang 1902, S. 243.

Mosso [33]. Experiments with the triceps suræ of the normal cat, i.e., without previous transection of the spinal cord, could only be done when the cat was under ether narcosis. The results obtained under

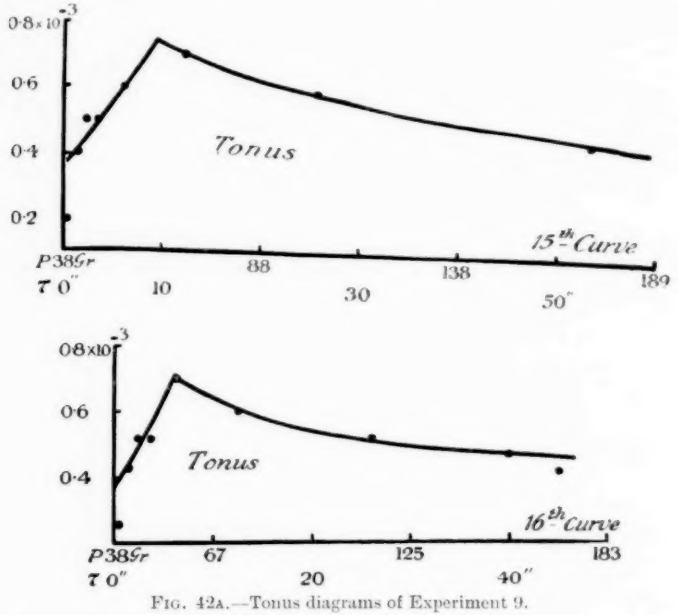

Fig. 42a.—Tonus diagrams of Experiment 9.

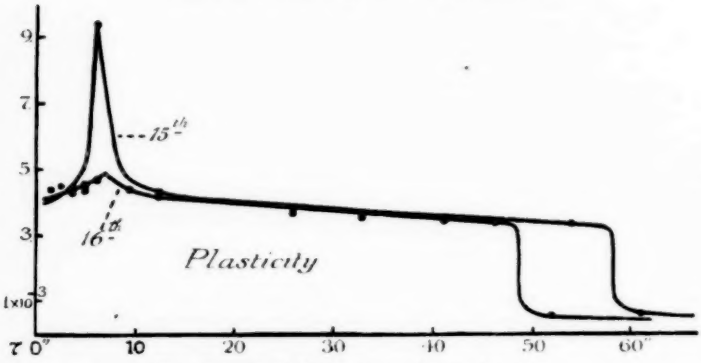

Fig. 43.—Plasticity diagrams of Experiment 9.

these experimental conditions are fully discussed in *Engelmann's Archiv*, Jahrgang 1902, and I will only quote here the conclusion. I concluded from these experiments *that probably the higher centres*,

and especially the cortex cerebri, exert an inhibiting influence upon the tonus of the skeletal musculature. The uncertainty introduced in these experiments by the narcosis made it desirable to perform some control experiments upon the normal human triceps suræ with the aid of Mosso's instrument. The curves which I obtained resemble those of cats when the narcosis is very superficial. The instrument of Mosso has the inconvenience that the curves made with it are deformed by

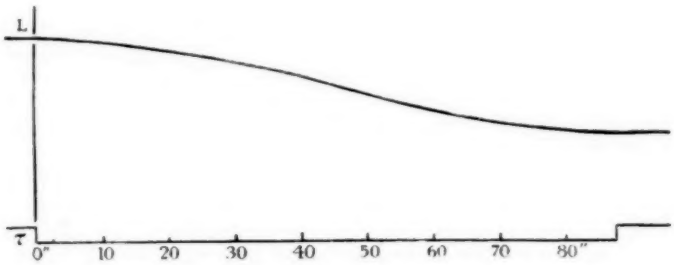

FIG. 44.—Length-load diagram of the tonic M. triceps suræ of L.

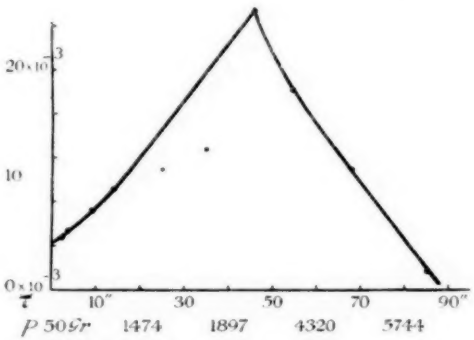

FIG. 45.—Tonus diagram derived from the extension curve of fig. 44.

respiratory movements. In order to meet this obstacle and to make the curves suitable for measurement, I copied the tracing, drawing the line through. In this way I eliminated, by a rather rough graphic method, the influence of the respiration upon the form of the myogram. This method introduces an element of subjectiveness into the experiment, but every other, even more refined, method would do the same. The extension curves all belong to the same type and resemble closely the curve published by Mosso and Benedicenti ([33], p. 356, fig. 3).

The values given in the tables represent by approximation the real increment of the load and the corresponding real increment of the length of the muscle. These values are obtained by multiplying the increment of the load on the scale-pan and the increment of the length

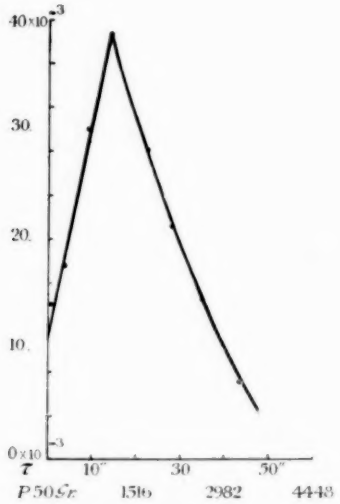

FIG. 46.—Tonus diagram of Experiment 11.

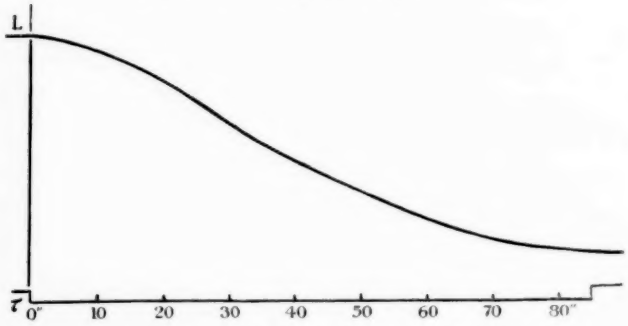

FIG. 47.—Length-load diagram of the tonic M. triceps suræ of B.

as recorded on the drum each by a coefficient deduced from the constants of the instrument and from the dimensions of the foot.

Experiment 10, *April* 6, 1902.—Triceps suræ of L. L. belongs, from the bodily aspect, to a hereditary hypotonic type (asthenicus,

Stiller). Fig. 44 is the copy of the original extension curve after elimination of the respiratory movements; fig. 45 is the derived tonus diagram. Table XLV contains the measurements.

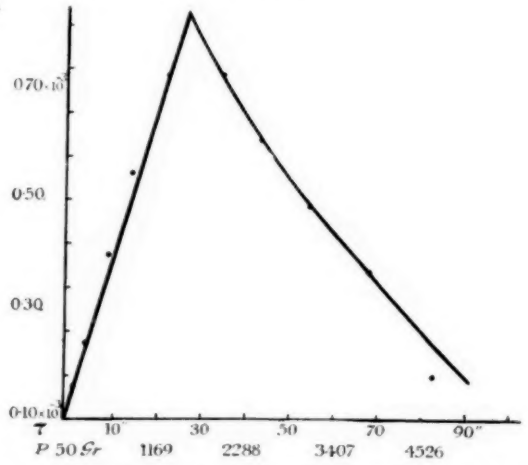

FIG. 48.—Tonus diagram derived from the extension curve of fig. 47.

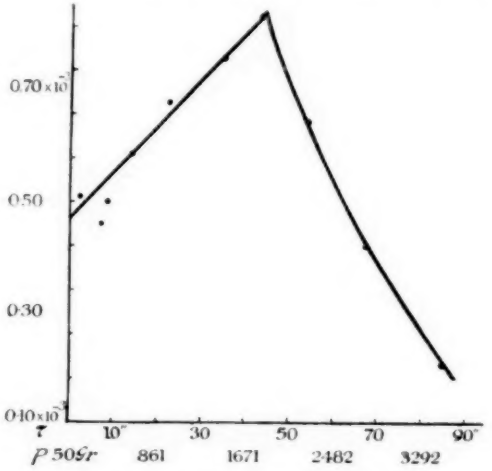

FIG. 49.—Tonus diagram of Experiment 13.

Experiment 11, *March* 16, 1902.—Triceps suræ of L. Fig. 46 is the tonus diagram, and Table XLVI contains the measurements.

Experiment 12, *April* 6, 1902.—Triceps suræ of B. B. belongs,

from the bodily aspect, to a hypertonic type, with several symptoms of vagotonia (Eppinger and Hess). Fig. 47 is the rectified extension curve, fig. 48 the derived tonus diagram. Table XLVII contains the measurements.

Experiment 13.—Triceps suræ of B. Fig. 49 is the tonus diagram, and Table XLVIII contains the measurements.

The characteristic feature of these tonus diagrams lies in the lengthening of the stage of increasing tonus and the shortening of the stage of decreasing tonus. The line representing the stage of increasing tonus is probably slightly curved in the beginning but tends very soon to a straight line. For the stage of decreasing tonus we find also in this case a logarithmic relation between the increment of the load and the decrement of the tonus. I found, when determining for this part of the tonus diagram the constants C_1 and C_2—

		C_1	C_2
L.	Experiment 10	$0{\cdot}15 \times 10^{-3}$	− 581
L.	,, 11	0·25	− 594
B.	,, 12	0·14	− 341
B.	,, 13	0·23	− 200

when approximately $\Delta = 0$. These constants are of the same order of magnitude as those in the spinal cat.

I must abstain from a further analysis of these extension curves, because my experimental material is not sufficient to trace further the rôle the elasticity and the plasticity play in the phenomenon of tonus, when the lower spinal centres are still under the control of the higher ones. We conclude, therefore, only *that the tonus diagram of the human triceps suræ resembles closely the tonus diagram of the same muscle in the normal cat when under superficial ether narcosis; that in comparison with the spinal cat the stage of increasing tonus is lengthened and the stage of decreasing tonus is shortened; that for the stage of decreasing tonus the logarithmic relation persists between the increment of the load and the decrement of the tonus.*

The experiments as yet described were all based upon the principle that the proprioceptors of a muscle are stimulated by stretching the muscle by means of an increasing weight. We may, however, also stimulate a proprioceptive reflex arc, at least its efferent part, by exciting the proprioceptors of the muscles antagonistic to those we wish to stimulate. The experiments based on this principle were exclusively

made with the frog. The skin over the pretibial muscles was opened and two electrodes, connected with an induction coil, were placed very carefully and without pressure upon the muscle group formed by the M. tibialis anticus longus and M. peroneus. The amount of the stimulus caused by the induced current was chosen so as to produce a strong continued contraction of the pretibial muscles. The experi-

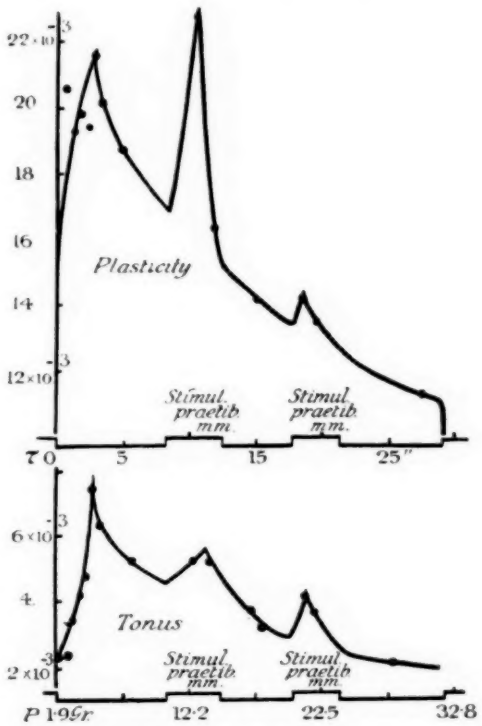

FIG. 50.—Tonus and plasticity diagrams of Experiment 14—fifteenth extension curve.

ment was performed in the following way. When making an extension curve of the M. gastrocnemius, I stimulated the pretibial muscles during a certain interval of time. I chose this form of the experiment because the stretching and extending of the M. gastrocnemius puts into action its own proprioceptive reflex arc, and under these circumstances the effect of the second stimulus caused by the faradization and the contraction of the pretibial muscles is more obvious.

Experiment 14 [*Table XIX, X, fig. 7*], *November* 29, 1899.—The frog used for the experiment was intact and wrapped up in cotton-wool. The pretibial muscles were stimulated by an induced current, the distance of the coils being 4·5 cm. The current induced by a distance of the coils of 8 cm. was hardly felt on the tongue, and at a distance of 3 cm. the current just caused pain on the tip of the tongue.

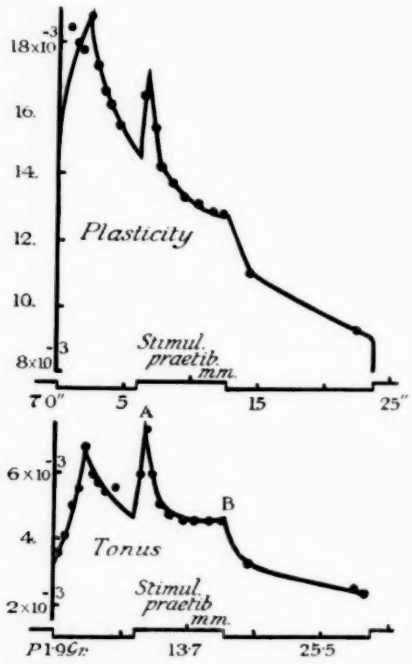

FIG. 51.—Tonus and plasticity diagrams of Experiment 14—sixteenth extension curve.

The pretibial muscles answered to this stimulation by a vigorous continued contraction. The stimulation lasted only a short while, viz., from 8·4 to 12·4 sec., and from 17·9 to 21·4 sec., reckoned from the beginning of the extension curve. Table XLIX contains the measurements of the fifteenth extension curve of the M. gastrocnemius, and fig. 50 represents the tonus and the plasticity diagrams. We see from this experiment that the stimulation of the proprioceptors of the pretibial muscles is followed by a strong increase of the tonus of the M. gastrocnemius. According to our definition of tonus, the M. gastro-

cnemius becomes more extensible, and this increment of the extensibility is chiefly caused by an increase of the plasticity of the muscle. The effect of the stimulation may still continue after the stimulus has already ceased. We conclude from this experiment *that the stimulation of the pretibial muscles by a faradic current, and the subsequent contraction of these muscles, cause a considerable increase of the tonicity of the M. gastrocnemius; this effect may even persist some seconds after the cessation of the stimulus. The increment of the tonus is chiefly due to an increase of the plasticity of the muscle.*

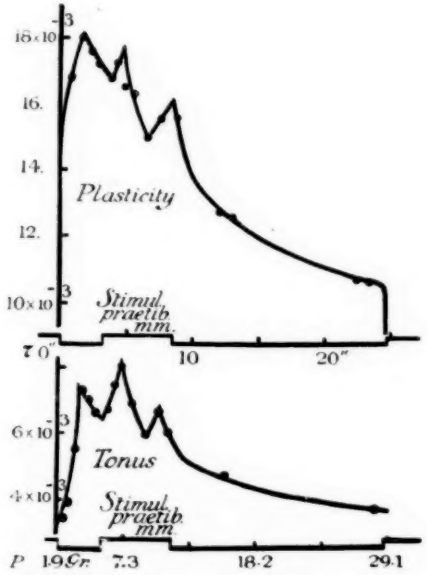

FIG. 52.—Tonus and plasticity diagrams of Experiment 15 (intact animal).

The increase in tonicity of the agonist under the influence of the contraction of the antagonist is usually called *inhibition* of the agonist by the antagonist. The rôle which these phenomena play in co-ordinate movement has been fully demonstrated by Sherrington and others.

Fig. 51 is the tonus and the plasticity diagrams of the next (sixteenth) extension curve of the M. gastrocnemius. The pretibial muscles were stimulated during the interval 5·9 to 12·8 sec., and by a distance of the coils of 4 cm. The stimulus was therefore, in this case, somewhat stronger and lasted longer than in the foregoing

experiment. The effect is a little different; the increment of the tonus is followed by a fall, which is again succeeded by a slight rise of the tonus. The curve representing the effect of the stimulation of the pretibial muscles upon the tonus of the gastrocnemius shows, therefore, two tops. Because these two tops will further be of interest for the description of the experiments, I will designate the first top as the A top, and the second one as the B top. The experiments now prove that the effect of the stimulation of the pretibial muscles is always a two-topped curve, provided the stimulus be of sufficient strength and of sufficient duration.

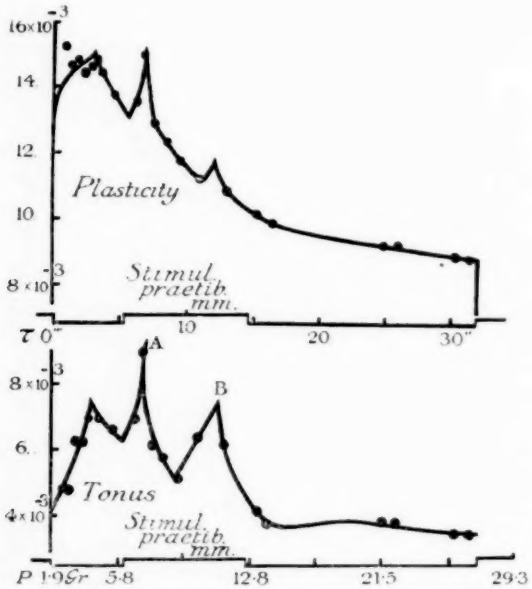

FIG. 53.—Tonus and plasticity diagrams of Experiment 15 (spinal cord transected).

Experiment 15 [*Tables XXVIII, XIX, fig. 8*], *November* 26, 1899.—This experiment was of the same kind as Experiment 14. The pretibial muscles, however, were on both sides carefully loosened from their insertions, and the distance of the coils was 5 cm. The loosening of the muscles from their insertions was done in order to avoid little movements in the joints and the concomitant stretching of the fasciæ, so that the effect of the stimulation was exclusively due to the excitation of the proprioceptors of the pretibial muscles.

Table L contains the measurements and fig. 52 the reproduction of the tonus and the plasticity diagrams. The effect of the stimulation is again a two-topped curve, and the persistence of the effect after the cessation of the stimulus is evident. In this case the B top is more marked than in the preceding experiment. The increase in the tonus always begins after a short period of latency.

Fig. 53 represents the tonus and the plasticity diagrams derived from an extension curve of the same frog, but after transection of the

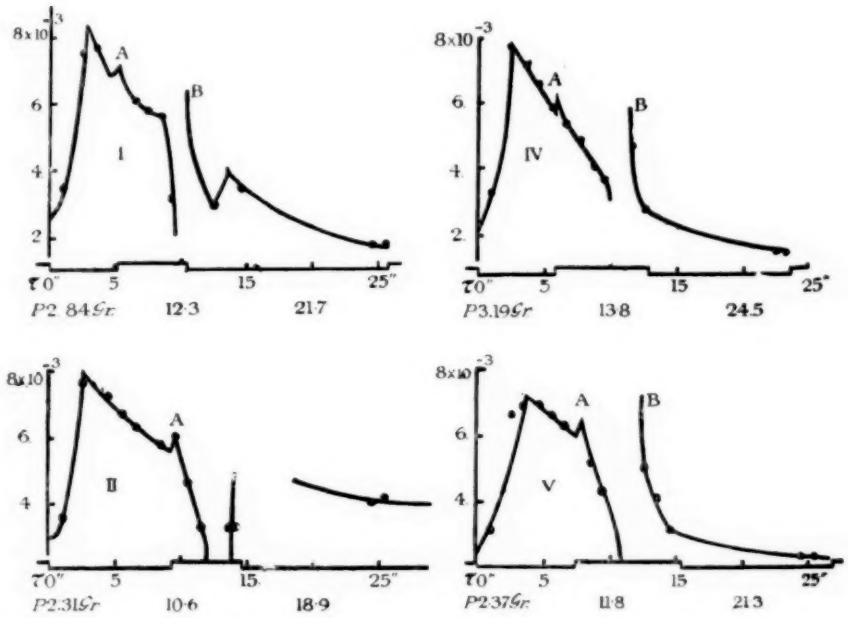

FIG. 54.—Tonus diagrams of Experiment 16.

spinal cord between the first and the second vertebræ. About one hour after the transection, the curve was recorded, being the eighth extension curve of the M. gastrocnemius after the transection of the cord. The effect is, in this case also, a two-topped curve. We conclude from these experiments that the stimulation of the pretibial muscles by a faradic current, and the subsequent contraction of these muscles, causes a considerable increase of the tonicity of the M. gastrocnemius. This increase of tonicity has two maxima, one shortly after the beginning of the excitation of the pretibial muscles (A top) and

another maximum in the neighbourhood of the cessation of the stimulus (B top). The second top is, however, not caused by the cessation of the stimulus, and it is only accidentally in these experiments that the B top falls in the neighbourhood of the cessation of the stimulus. The moment at which the second top appears seems only to be determined by the strength of the stimulus and the actual state of the reflex centre.

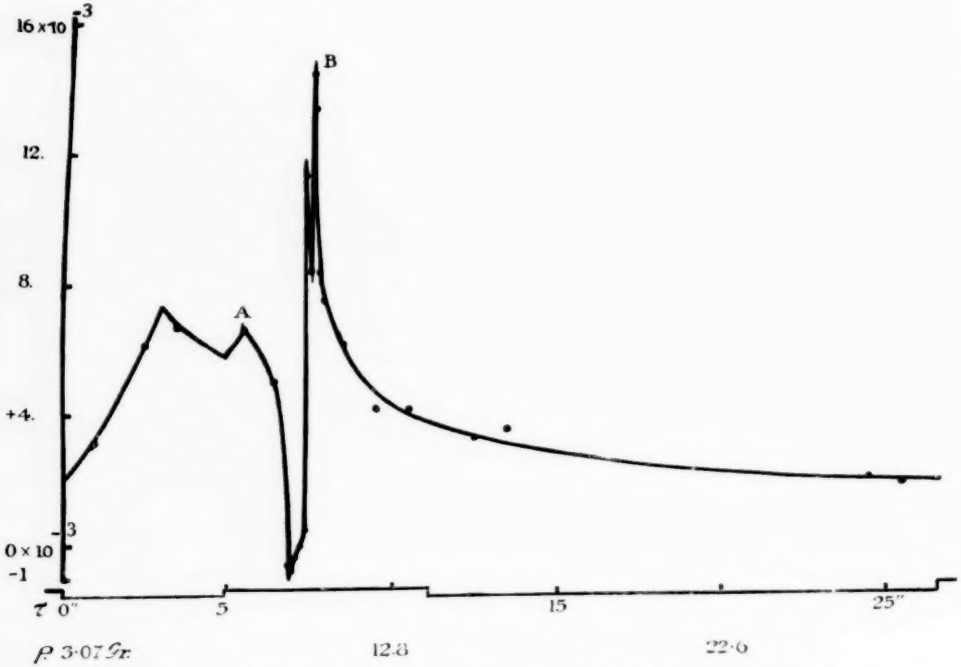

FIG. 55.—Tonus diagram of Experiment 16—third curve.

Experiment 16, *November* 25, 1899.—This experiment belongs to the same group as Experiments 14 and 15. The frog was intact, and wrapped up in cotton-wool. The pretibial muscles were loosened on either side from their insertions to avoid little movements in the joints. I reproduce in figs. 54 and 55 the tonus diagrams derived from five successive extension curves of the M. gastrocnemius. The first of these extension curves is about the fortieth curve of the whole series, recorded with intervals of approximately five minutes.

First curve (fig. 54, I).—The pretibial muscles were stimulated during the interval 5·3 to 10·4 sec., reckoned from the beginning of the extension curve, the distance of the coils being 4 cm. The initial charge amounted to 2·84 grm. and the increment of the charge to 0·94 grm. per second.

Second curve (fig. 54, II).—Interval of stimulation 8 to 14·6 sec.; distance of the coils 4 cm.; initial charge 2·31 grm.; and increment of the charge 0·83 grm. per second.

Third curve (fig. 55).—Interval of stimulation 4·9 to 11·1 sec.; distance of the coils 4 cm.; initial charge 3·07 grm.; and increment of the charge 0·98 grm. per second.

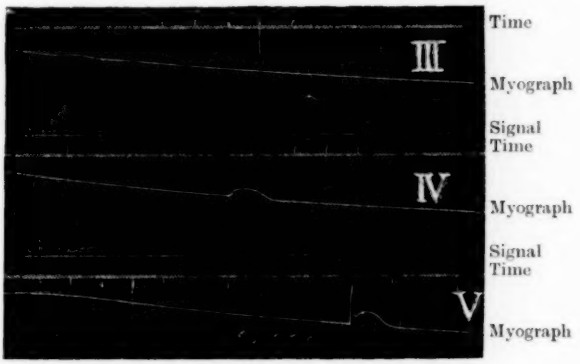

FIG. 56.—Three successive extension curves of the tonic M. gastrocnemius. The curves marked III and IV show a tonic contraction evoked by the stimulation of the pretibial muscles. The curve marked V exhibits at the same time a twitch and a tonic contraction. The dropping of the signal indicates the moment at which the pretibial muscles were stimulated. The enlargement is about ten times. The time in tenths of seconds.

Fourth curve (fig. 54, IV).—Interval of stimulation 5·9 to 12·8 sec.; distance of the coils 4 cm.; initial charge 3·19 grm.; and increment of the charge 1·07 grm. per second.

Fifth curve (fig. 54, V).—Interval of stimulation 7·4 to 15·4 sec.; distance of the coils 4 cm.; initial charge, 2·37 grm.; increment of the charge 0·95 grm. per second.

Fig. 56 is a reproduction of the original extension curves from which the third, fourth, and fifth tonus diagrams are derived.

The five extension curves of this experiment exhibit the same general feature as those already described, but about four seconds

after the beginning of the stimulation of the pretibial muscles a slight contraction appears. Jäderholm [24], who gives an ample description of this kind of contraction, designates it by the very appropriate name of "tonic contraction." The tonic contraction and the twitch are the two different forms of contraction of which the striped muscle is capable. These two forms of contraction are independent of each other, as may be seen from Curve V, fig. 56, which shows a twitch and a tonic contraction at the same time. The twitch is, without doubt, connected with the function of the striped apparatus, and therefore under the control of the motor cell of the anterior horn. The tonic contraction, however, seems to my opinion connected with the sarcoplasmatic mass, and hence controlled by the sympathetic motor cell, which innervates this part of the muscle.[1]

An interpolation shows that the twitch begins about one-tenth of a second after the commencement of the tonic contraction. The same result was found in other cases, so that we conclude *that the tonic contraction in its appearance precedes the twitch for about one-tenth of a second.* A tonic contraction of the M. gastrocnemius is more easily obtained by stimulating the proprioceptors of the pretibial muscles than a twitch, and only a repeated or continued stimulation of sufficient strength is able to elicit a twitch. We conclude from these observations *that the impulses set up in the afferent part of a proprioceptive reflex arc by stimulating the proprioceptors of a muscle not only go over upon the efferent part of its own proprioceptive reflex arc, but also upon the efferent part of those reflex arcs, which belong to muscles antagonistic to the stimulated one. The way of least resistance for the passage of the stimulus to the efferent part of an antagonistic reflex arc leads to the sympathetic motor cell.* Upon this principle is built the interaction of antagonistic muscles in the production of co-ordinated movements.

The tonic contraction may be considered as a sudden variation of the tonus, as illustrated by the tonus diagram of fig. 55. This diagram is exactly of the same form as the tonus diagrams reproduced in figs. 50, 51, 52 and 53, the only difference being that the fall of the tonus between the two tops is deeper. The difference between the diagrams, in which a tonic contraction appears, and those in which this is not the case resides, therefore, only in the depths of the tonus fall, occurring between the A and B tops. Hence we conclude that the cases in which

[1] Botazzi and Grünbaum [12] arrived at a similar result for the auricular musculature of the tortoise heart [12].

a tonic contraction appears are continuously connected with those in which this is not the case, or, in other words, *that the tonic contraction is only a sudden and considerable variation of the tonus.*

The tonus diagram of fig. 55 shows a peculiarity, viz., an accessory top between the A and B tops. In order to know if this accessory top

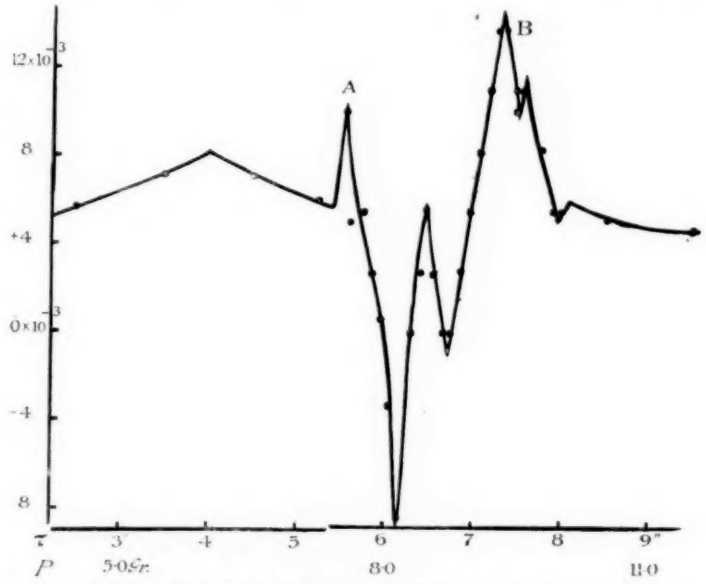

FIG. 57.—Tonus diagram belonging to Experiment 16, and showing accessory tops.

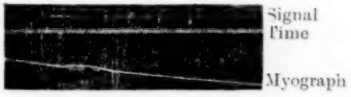

FIG. 58.—Extension curve of the tonic M. gastrocnemius, showing a tonic contraction evoked by the stimulation of the pretibial muscles. The dropping of the signal indicates the moment at which the pretibial muscles were stimulated. The enlargement is about ten times. The time in tenths of seconds.

was due to an accidental irregularity in the original tracing, I took another extension curve of the M. gastrocnemius, belonging to the same series, and also showing an apparently smooth tonic contraction. Table LI contains the measurements of this curve, and fig. 57 is the tonus diagram. The original curve is reproduced in fig. 58; there is also in this case an accessory top between the A and B tops, and

above them little accessory tops upon the descending branch of the B top. The same phenomenon shows the tonus diagram of fig. 59, which is derived from the extension curve reproduced by fig. 60. This tracing belongs to another series, and the stimulation of the pretibial muscles took place at a distance of the coils of 6 cm. At first sight the tonic contraction seems smooth and continuous, but an accurate measurement of the curve reveals three accessory tops between the A and B tops. The

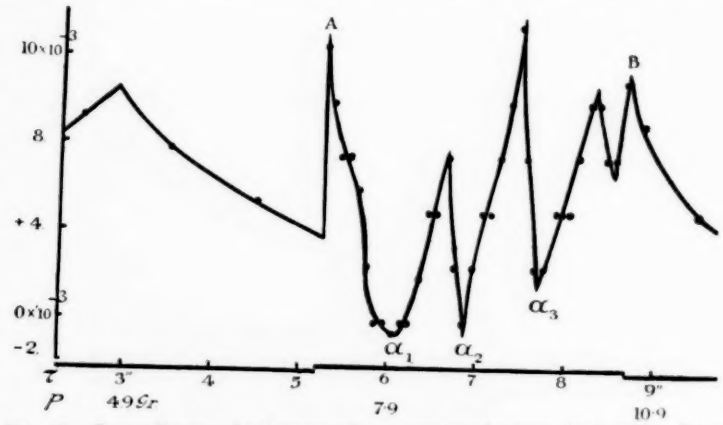

FIG. 59.—Tonus diagram showing several accessory tops between the A and the B top.

FIG. 60.—Extension curve of the tonic M. gastrocnemius showing a tonic contraction evoked by the stimulation of the pretibial muscles. The dropping of the signal indicates the moment at which the pretibial muscles were stimulated. The enlargement is about ten times. The time in tenths of seconds.

three tops a_1, a_2, a_3, in the tonus diagram are approximately equidistant, viz., 0·75 sec., and this fact suggests the idea that the tonic contraction is a tetanus, even in those cases in which it has the appearance of a continuously smooth contraction. That this is really the case is demonstrated by Curve IV of fig. 56. The ascending branch of the tonic contraction shows clearly a staircase phenomenon. Fig. 61 shows the same phenomenon in a more marked way; this curve is the first extension curve of the series of five, of which fig. 56 reproduces the third, fourth and fifth tracings. Not only the ascending branch

but also the descending one may exhibit the staircase phenomenon. An example of this kind shows fig. 62; this curve is the second one of the same series of five. The fact that the descending branch may also exhibit the staircase phenomenon is a further proof of the tetanic nature of the tonic contraction. We conclude, therefore, from these experiments *that the tonic contraction of the M. gastrocnemius evoked by stimulating the pretibial muscles by means of a faradic current is not a single contraction but a tetanus.*

After the tonic contraction has passed away the muscle often remains oscillating. The amplitude of the oscillation is extremely small, hardly perceptible in the tracing, notwithstanding the enlargement of the movement by the length-recorder is about tenfold. The duration of the oscillations is approximately constant, but may differ from case to case. The variation in the duration of the oscillations is about 1·5 to 2·5 sec.[1]

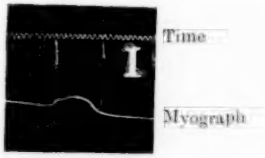

FIG. 61. — Tonic contraction of the M. gastrocnemius. The ascending branch of the contraction shows a staircase. The enlargement is about twenty times. The time in tenths of seconds.

FIG. 62. — Tonic contraction of the M. gastrocnemius. The ascending as well as the descending branch shows a staircase. The enlargement is about twenty times. The time in tenths of seconds.

Curve III, fig. 56, shows four of these oscillations, beginning shortly after the tonic tetanus has finished. The duration of each oscillation is about 1·6 sec.

The curves representing the tonic contraction of the M. gastrocnemius of the frog resemble closely the contraction curves of smooth muscles published by Engelmann, Winkler, Schultz, and others. The tetanic character of the tonic contraction, the staircase phenomenon, the oscillations following the tonic tetanus, the slowness of the contraction and its little height are all the same. I conclude, therefore, *that the tonic contraction and the phenomena allied with it are due to the sarcoplasmatic part of the striped muscle. Hence we come from the physiological side also to the conception of the duality of the striped muscle, viz., a*

[1] The same oscillatory movements are described by Ph. Botazzi and others in the case of smooth muscles of Evertebrata [11] and [12].

sarcoplasmatic mass innervated by a sympathetic motor cell, which in its function closely resembles the smooth muscles and a striped apparatus, termination of the motor cell of the anterior horn, embedded in this sarcoplasmatic stroma.

Keith Lucas [29], in his experimental work, arrived at a similar result, viz., that the muscle is composed of three different contractile substances. In my opinion his general conclusion that there are different contractible substances in the striped muscle is true, but my experiments tend to the conclusion that there are only two, in agreement with the morphological researches of Boeke.

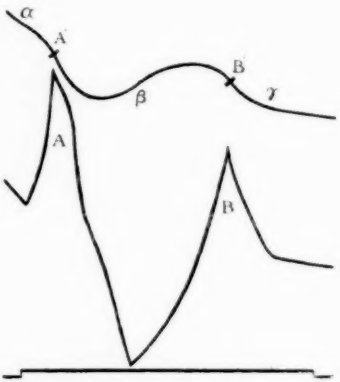

FIG. 63.—Scheme representing the effect of the stimulation of the pretibial muscles upon the form of the extension curve of the M. gastrocnemius. The curve $a\beta\gamma$ is the extension curve, and the curve AB the derived tonus diagram.

The curve representing the effect of the stimulation of the pretibial muscles upon the extensibility of the M. gastrocnemius is **S**-shaped. Fig. 63 represents this effect diagramatically. The tonus diagram AB, derived from the extension curve $a\beta\gamma$, is constructed on the supposition that the load augments uniformly as in our experiments. The temporary lengthening of the muscle represented by the part $a\beta$ is usually called "inhibition," and the subsequent shortening of the muscle "tonic contraction." Inhibition and tonic contraction are two phases of the same process evoked by the stimulation of the proprioceptors of an antagonistic reflex arc. A good instance of such an **S**-shaped curve shows fig. 60.

The form of the $a\beta\gamma$ curve is extremely variable in different experi-

ments; it is determined by the anatomical structure of the reflex centre, by the actual state of this centre, and by the nature and the strength of the stimulus. The point *a* indicates the moment at which the inhibition begins. The determination of the exact position of this point upon the curve is only possible in the tracings of figs. 60, 64 and 65. In these cases the latent period between the beginning of the stimulation and the beginning of the inhibition varies between 0·05 and 0·1 sec. The A top of the tonus diagram coincides with the first inflection point [A′] of the extension curve, and the B top with the third inflection point [B′], situated upon the descending branch of the tonic contraction. The point *β* indicates the moment at which the tonic contraction begins. This point may easily be determined in the tracings. The five successive tracings belonging to Experiment No. 16 give the following result:—

Number	Moment at which the stimulation of the pretibial muscles begins	Beginning of tonic contraction	Difference in seconds
I	5·3	9·7	4·4
II	8·0	12·0	4·0
III	4·9	6·8	3·9
IV	5·9	9·9	4·0
V	7·3	10·5	3·2

These five experiments were made under similar conditions, and repeated with intervals of about five minutes and with the same strength of stimulus. We see from this table that the tonic contraction begins about four seconds after the commencement of the stimulation of the pretibial muscles, but that there are slight deviations from this mean in accordance with the reflex nature of the whole phenomenon. In the same way the point may be determined at which the tonic contraction has ceased. The table gives the relation between the end of the tonic contraction and the cessation of the stimulus:—

Number	End of the tonic contraction	Cessation of stimulation of the pretibial muscles	Difference in seconds	Duration of the tonic contraction in seconds
I	10·9	10·4	− 0·5	1·2
II	13·4	14·6	+ 1·2	1·4
III	8·0	11·1	+ 3·1	1·2
IV	11·4	12·8	+ 1·4	1·5
V	12·2	15·4	+ 3·2	1·7

It follows from this table that there is no fixed relation between the end of the tonic contraction and the moment at which the stimulus

ceases. The duration of the tonic contraction is, however, approximately constant. Other experiments give similar results. Hence we conclude *that the effect following the stimulation of the pretibial muscles is chiefly determined by the actual state of the reflex centre and by the strength of the stimulus, but that the duration of the stimulation has but little influence*, in other words, *that the effect of the stimulation may be regarded as a reflex discharge.*

The inhibition of the tonus lasts about three times as long as the tonic contraction, and when the strength of the stimulus increases, the inhibitory effect becomes more and more predominant. Fig. 64 may prove this. The curve belongs to the series of Experiment 16 and was the thirteenth tracing. The distance of the coils amounted to but

FIG. 64.—Extension curve of the tonic M. gastrocnemius showing a tonic contraction evoked by the stimulation of the pretibial muscles. The dropping of the signal indicates the moment at which the pretibial muscles were stimulated. In this case the inhibitory effect is predominant. The enlargement is about ten times. The time in tenths of seconds.

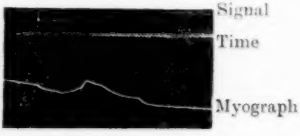

FIG. 65.—The same experiment as reproduced in fig. 64, but with a still stronger stimulus. The enlargement is about ten times. The time in tenths of seconds.

3 cm., so that the stimulus in this case was rather strong. When the strength of the stimulus increases still further, the general feature of the curve remains the same, viz., **S**-shaped, but the tracing becomes irregular. Fig. 65 may prove this. The curve of fig. 65 is the fourteenth of the same series, and the distance of the coils amounted to 2·5 cm. We conclude from the preceding experiments *that inhibition of tonus and tonic contraction are two phases of the same excitatory process.* The inhibitory effect is only caused by the fact that the sarcoplasmatic part of the striped muscle shortly after the beginning and also near the end of its excitatory state shows an increment of its extensibility, which is due to a temporary increase of its plasticity.[1] The contraction curve of smooth muscles shows also two maxima of extensibility, one shortly after the beginning of the contraction and the other near the end, exactly in the same way as the tonic contraction

[1] Schenck also established the fact that the extensibility of the striped muscle is increased at the beginning of the contraction (*Pfluger's Arch.*, 1900, Bd. lxxxi, S. 595).

of the striped muscles. *There exists also a close analogy between the function of the smooth muscle and the function of the sarcoplasmatic part of the striped muscle* [23].

Chapter III.—The Isotonic Contraction.

The analysis of the extension tracings of the tonic muscle in the second section of this research leads to the conclusion that the formula

$$-l = \frac{1}{C_1 C_2}\left[C_1(P-\Delta) - \lg n.C_1(P-\Delta) - 1\right]$$

represents the shortening of the muscle [$-l$] caused by the stimulation of the proprioceptors through a uniformly increasing weight [P]; this result, however, could not be tested directly. I tried, therefore, to prove that the formula quoted above represents the relation between the amount of the stimulus in physical units and the lift of the muscle when working under isotonic conditions. The conditions under which the muscle is contracting in the two cases are not strictly the same, but these differences will only affect the value of the constants of the formula, not the form of the function. Much work has been done by the earlier observers to elucidate the relation between stimulus and lift for a muscle contracting isotonically. A great deal of this work was performed by very skilful experiment, and in such an exact way that it seems for my purpose superfluous to repeat it. Hence I shall use in this section the experimental material of others.

The problem involves in the first place the question of the relation between the stimulus in physical units and the stimulus in physiological units, or whatever part of the stimulus, considered as a physical cause, is transmuted into a physiological cause. Generally it is admitted that the relation

$$\frac{\text{stimulus in physical units}}{\text{stimulus in physiological units}} = \text{constant.}$$

Now I have shown in my publication on the principle of entropy in physiology [28] that all physiological systems are endowed with passive resistances, so that the increment of the stimulus ought to surpass a certain finite limit before producing an increment of the effect. We find, therefore, that all the stimuli [$P \pm \delta$], in which [δ] means a finite increment of P, yield the same effect. The value of [δ] may be comparatively great when the passive resistances in the physiological system are considerable. It is evident from the simple recognition that all

physiological systems are systems with passive resistance that the same relation

$$\frac{\text{stimulus in physical units}}{\text{stimulus in physiological units}} = \text{constant}$$

cannot exist for the whole scale of stimuli. For though $[\delta]$ is small in general in comparison with P, it will nevertheless sensibly influence the value of P when P itself is small. Hence we conclude *that if we apply, as is usually done, the relation*

$$\frac{\text{stimulus in physical units}}{\text{stimulus in physiological units}} = \text{constant}$$

to the whole scale of stimuli, we ought to find deviations between the observed and the calculated values of $[-1]$ *in the neighbourhood of the small values of* P.

The second question involved in the problem ensues from the plasticity of the muscle. As I have proved in the first and second sections, the muscle, in consequence of its plasticity, is easily deformed by mechanical and thermical agencies. The same happens when the muscle is compelled to contract by electrical or other artificial stimuli, applied to the motor nerve. After each contraction the muscle is slightly deformed, and when this deformation surpasses a certain limit, the effect becomes manifest in the form of a slight permanent shortening of the muscle. This deformation was first systematically studied by Tiegel [43], and later more in detail by Bohr [10]. This fact is of the highest interest, because it shows that in a series of successive contractions the muscle does not remain the same object. Hence *it is impossible to represent the relation between stimulus and lift by a formula in which enters only one independent variable—viz., the stimulus.* A formula which will give a true representation of the relation between the stimulus and the lift ought to contain at least two independent variables—viz., the variable P, the stimulus, and a second variable Q, which represents the momentary state of the muscle during the experiment; or, in other words, the effect of the stimulation is not entirely determined by the value of the stimulus in physical units. Hence we conclude *that it is only possible to represent the relation between stimulus and lift by a formula containing one independent variable, if we neglect the plastic deformation of the muscle during the experiment,* or if we try to eliminate by some artifice the influence of the plastic deformation upon the effect of the stimulation.

The formula

$$-1 = \frac{1}{C_1 C_2} \left[C_1 (P - \Delta) < \lg n.\ C_1 (P - \Delta) - 1 > \right]$$

is reduced to its simplest form when we substitute $C_1 (P - \Delta) = R$. After this substitution it assumes the form

$$- l [C_1 C_2] = F(R) = R \{ M \log. R - 1 \},$$

if M is the modulus. Fig. 66 is the graphic representation of the function $F(R) = R \{ M \log. R - 1 \}$. The first striking feature of this function is that it shows a maximum in the neighbourhood of R = 76, and it may be asked if this agrees with the physiological facts. The opinions about this point are divided, and a great many observers, such as Tigerstedt, believe that the lift approaches asymptotically a limiting value, when the stimulus increases. In my opinion this conclusion is erroneous, because it is left out of consideration that the muscle is so

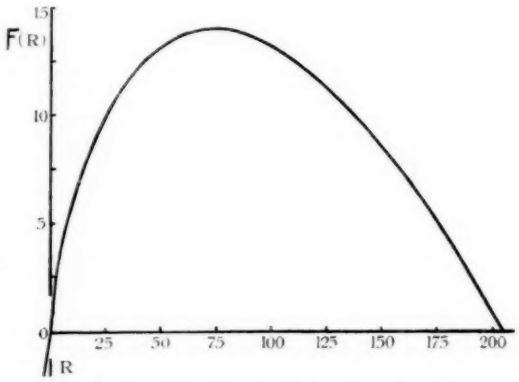

FIG. 66.—Graphic representation of the function $F(R) = R \{ M \log. R - 1 \}$.

easily deformed by a strong stimulus. It seems, therefore, to me that the asymptotical increment of the lift is not caused by a further real increase of the lift, but only by a shortening of the muscle, caused by plastic deformation. I agree, therefore, with Waller and others, who believe that the lift considered as a function of the stimulus is a maximum-phenomenon. It is another question, whether the decrement of the function by a further increment of R is also realized in nature. The experiments seem to indicate that this is really the case, but that the decrement of the lift by further increase of the stimulus in most cases is hidden by the plastic deformation of the muscle.

Experiment 20, *of Tigerstedt and Willhard*, proves the existence of a maximum lift and shows, moreover, the plastic deformation of the

muscle. For this experiment a muscle-nerve preparation of the frog is used. The nerve is stimulated by a break-induction shock. First the stimulus increases, and as soon as it reaches the value of 70, the maximum lift of 13·25 is attained; then the stimulus is gradually augmented to 75 and to 80, but the lift is only 13·15. By a further increase of the stimulus the plastic deformation becomes evident, and

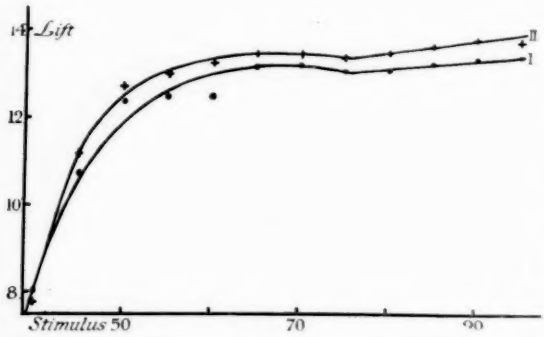

FIG. 67.—Graphic representation of Experiment 20 of Tigerstedt and Willhard. The lift as a function of the amount of the stimulus in arbitrary physical units.

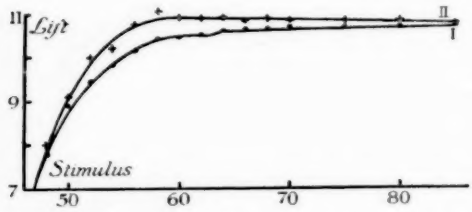

FIG. 68.—Graphic representation of Experiment 28 of Tigerstedt and Willhard. The lift as a function of the amount of the stimulus in arbitary physical units.

the lift seems to increase. When the stimulus has attained the strength of 95, the course of the experiment is reversed, and the stimulus decreases by equal steps, in the same way as it had previously increased. The curves representing the lifts as a function of the amount of the stimulus in arbitrary physical units are reproduced in fig. 67. Curve I represents the lifts by increasing stimuli, and Curve II those by decreasing stimuli. It is evident that the curves coincide at the beginning and

diverge towards the end, and that the second curve lies above the first. This proves that the muscle is temporarily deformed by the supramaximal stimuli.

Experiment 28 *of Tigerstedt and Willhard* is of the same kind, and shows the same phenomena (fig. 68). The second curve is remarkable, because during the interval of supramaximal stimuli the lifts at first increase by decreasing stimuli. A better proof that the muscle is deformed by strong stimuli could hardly be given. The lines representing the relation between stimulus and lift for supramaximal stimuli are approximately straight ones, and this harmonizes exactly with the results obtained by Bohr [10] during tetanus of the muscle.

I used the following method to prove the agreement between the formula and the observations. For $R = C_1(P - \Delta) = 0$, the value of the function, or $-l\,[C_1C_2]$, is also zero; this means that the stimulus in physical units, P, will have no visible effect until P surpasses the threshold value Δ. Now I plotted the observed values of $-l$ against the values of P and extrapolated this curve graphically. In this way Δ is fixed. By means of Δ the value of the stimuli $(P - \Delta)$ is determined. Next I tried to determine graphically the top of the tracing. In choosing the experiments, where this was possible, I eliminated those in which the plastic deformation was very dominant. At the top of the curve R is approximately 76, and I pose, therefore, $R_{max.} = 76$ $C_1 (P_{max.} - \Delta)$. $P_{max.}$ means the value of P in physical units, for which the lift is a maximum. By the determination of the top, $P_{max.}$ is also fixed, and hence $C_1 = \frac{76}{P_{max.} - \Delta}$. Because P is given in arbitrary units, the determination of C_1 is always possible. C_1 once fixed in this way, the value of the stimulus $C_1 (P - \Delta)$ may be determined for the whole scale of stimuli. I should propose to call this method the method of the equivalent stimuli, because it assumes that those stimuli are physiologically equivalent which produce the maximum lift. I will not enter upon a discussion of the intrinsic value of this proposition, because it is only applied here for the sake of convenience to have an easy method of testing the formula. When the reduced stimuli $C_1 (P - \Delta)$ have been calculated, we may also determine the value of the function

$$-l[C_1C_2] = C_1 (P - \Delta) \{ M \log. C_1(P - \Delta) - 1 \}.$$

The top of the curve agrees with $C_1 (P - \Delta) = 76$, and at that moment the value of the function is approximately 13·98; hence I pose—

$$L_{max.} = 13{\cdot}98 = l_{max.}[C_1C_2], \text{ or}$$

$\frac{13{\cdot}98}{l_{max.}} = C_1C_2$; $l_{max.}$ means the value of l at the top or the maximum lift. By means of this relation C_2 is calculated. There are other methods to test the agreement between the formula and the observed values of l, which are equally simple, but by the method of the equivalent stimuli the influence of the plastic deformation is for a great deal eliminated.

(I) *Experiments of Tigerstedt and Willhard*, 1883 [44]. — These observers used a muscle-nerve preparation of the frog. The nerve was stimulated by electrical stimuli.

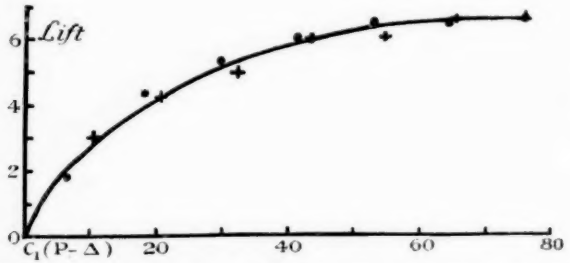

FIG. 69.—Graphic representation of Experiment 6 of Tigerstedt and Willhard. The lift as a function of the reduced stimuli.

Experiment 6.—The nerve is excited by make-induction currents. The mean intensities of the induced currents are proportionate to the numbers given in the second column of Table LII. The stimuli in physical units, P, are therefore proportionate to the mean intensities of the induction currents. Because the mean velocity with which the current increases is approximately independent of the intensity, it is only this last quantity which forms the independent variable of the experiment. The stimulations of the nerve succeeded each other at equal intervals of eight seconds. In the third column the reduced stimuli are given; in the fourth row the lift as observed by Tigerstedt and Willhard; in the fifth column the value of the lift calculated by means of the constants of the sixth column. Fig. 69 is the graphic representation of Table LII. The curve of this figure represents the lifts as a function of the reduced stimuli, calculated by means of the constants of the sixth row. The observed values of the lift are indicated by dots when belonging to a series of increasing stimuli, and by crosses when belonging to a series of decreasing

stimuli. In this experiment the two curves calculated by means of the method of the equivalent stimuli coincide.

In order not to burden this part of my research, I will publish the results in as condensed a form as possible. Hence it may suffice to mention only the fact that Experiment 27A of Tigerstedt and Willhard gives the same result without publishing the numerical data. The two curves approximately coincide, and the agreement between the observed and the calculated values of the lift is of the same kind as in the preceding experiment.

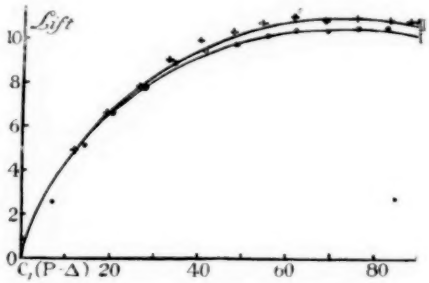

FIG. 70.—Graphic representation of Experiment 28 of Tigerstedt and Willhard. The lift as a function of the reduced stimuli.

Experiment 28 of Tigerstedt and Willhard.—This experiment is of the same nature as the preceding one. The nerve is stimulated by break induction shocks. Table LIII contains the numerical results, and fig. 70 is the graphic representation of the experiment. In this case the two curves do not coincide. The curve marked I, belonging to the series of increasing stimuli, lies below the curve marked II, pertaining to the series of decreasing stimuli. The curves coincide at the beginning and diverge towards the end. This fact proves that a long sequence of increasing stimuli, and especially the stronger and supramaximal stimuli, have temporarily deformed the muscle. This experiment shows also clearly the deviations between the observed and the calculated values of the lift for small values of P.

Experiment 20 of Tigerstedt and Willhard shows the same facts, and the agreement between the observed and the calculated values of the lift is the same.

Experiment 38 of Tigerstedt and Willhard.—In this case the nerve was stimulated by the make and break of a constant current of increasing intensity. Also in this case the mean velocity, with which

the current increased or decreased, was approximately constant, independently of the intensity of the constant current. Table LIV and fig. 71 contain the results of the experiment.

Experiment 32A *of Tigerstedt and Willhard* (Table LV, fig. 72).—In this experiment the nerve was curarized and the muscle stimulated directly with break-induction currents. In this case also there exists agreement between the observed and the calculated values of the lift·

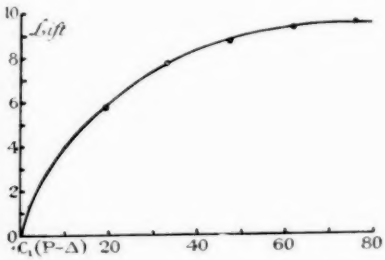

FIG. 71.—Graphic representation of Experiment 38 of Tigerstedt and Willhard. The lift as a function of the reduced stimuli.

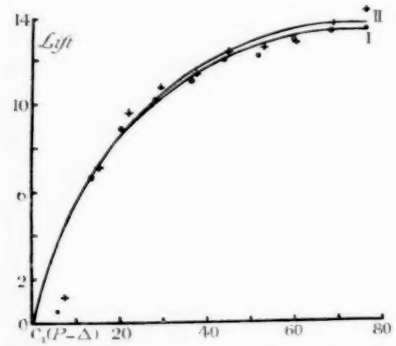

FIG. 72.—Graphic representation of Experiment 32A of Tigerstedt and Willhard. The lift as a function of the reduced stimuli.

The deviation for small values of P is very marked. Experiment 33A of the authors is of the same kind, and yields similar results.

(II) *Experiments of von Kries* [27] 1884.—von Kries worked with the M. gastrocnemius of the frog after destruction of the medullary cord. The nervus ischiadicus is stimulated by a uniformly increasing current (Feder-rheonom). The velocity of the increment of the current

was constant throughout and only the intensity varied. The numbers in the second column are proportionate to the intensities of the exciting current. Hence there is only one independent variable in the experiment.

Experiment 2 of von Kries (Table LVI, fig. 73).—The nerve was excited by a current which reached its maximum intensity in about 0·05 second.

Experiments 1 and 3 give the same result.

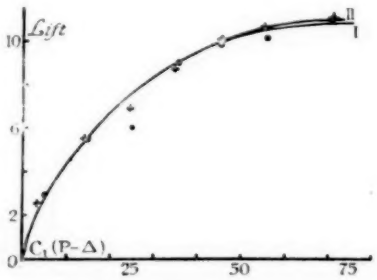

FIG. 73.—Graphic representation of Experiment 2 of von Kries. The lift as a function of the reduced stimuli.

(III) *Experiments of Cybulski and Zanietowski* [16], 1894.—The experiments of the second isotonic series are only suited to my purpose. The authors used the gastrocnemius nerve preparation of the frog. The nerve is stimulated by means of condenser discharges; the capacity of the condenser is 1×10^{-8} F. The charge of the condenser was varied. It may be concluded, from the statements made by the authors about their apparatus, that the mean intensity of the discharge current increases approximately proportionately to the charge of the condenser. The velocity of the increment of the discharge current is but slightly affected by the variation of the mean intensity, so that also in this case there is only one independent variable, which is proportionate to the charge of the condenser. The stimulus in physical units is therefore expressed in coulombs in agreement with the authors.

Experiment 3 of Cybulski and Zanietowski, with omission of the first ten observations (Table LVII, fig. 74). This experiment is remarkable for the fact that it shows so very clearly the deformation of the muscle by supramaximal stimuli. The Curve AB represents the relation between the stimulus in physical units and the lift; Curve BC is the continuation

of the experiment. It represents the "lifts" for the supramaximal stimuli. It seems evident to me that in a continuously proceeding experiment, the Curve AB cannot represent the same phenomenon as the Curve BC. It is obvious that the Curve BC is not the direct continuation of the Curve AB and that this Curve BC does not approach

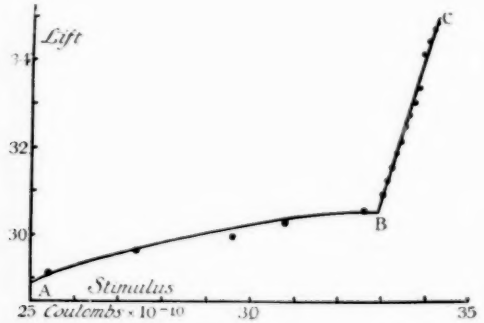

FIG. 74.—Graphic representation of Experiment 3 of Cybulski and Zanietowski. The lift as a function of the amount of the stimulus in arbitrary physical units.

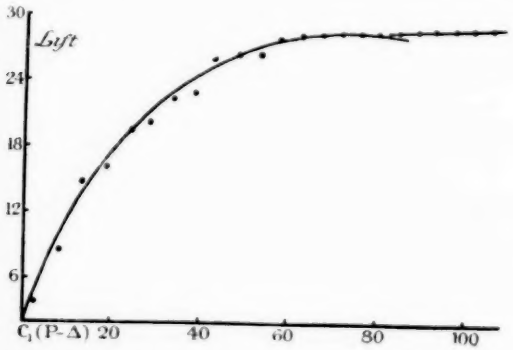

FIG. 75.—Graphic representation of Experiment 2 of Cybulski and Zanietowski. The lift as a function of the reduced stimuli.

asymptotically any limiting value. The Curve BC is rectilinear in the same way as in the experiment of Tigerstedt and Willhard, and represents the deformation of the muscle by supramaximal stimuli.

Experiment 2 of the same authors (Table LVIII, fig. 75) proves the agreement between the observed and the calculated values of the lift. The top of the curve lies in the neighbourhood of P = 31·9, and from

that point on the divergence begins. The line uniting the "lifts" caused by the supramaximal stimuli is a straight one also in this case.

Experiments 6A *and* 6B.—Both experiments are made with the same preparation and under the same experimental conditions. Experiment 6A precedes Experiment 6B. The charge of the muscle in Experi-

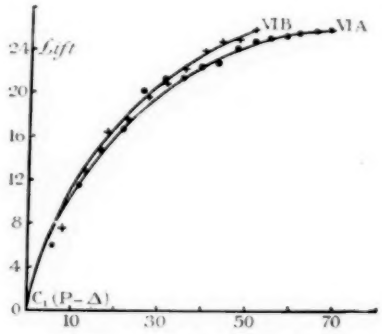

FIG. 76.—Graphic representation of Experiments 6A and 6B, of Cybulski and Zanietowski. The lift as a function of the reduced stimuli.

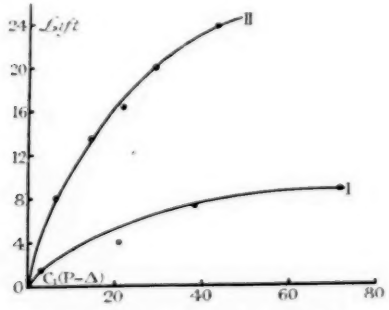

FIG. 77.—Graphic representation of an experiment of Cybulski and Zanietowski. The lift as a function of the reduced stimuli. Curve I twitches, Curve II tetani of short duration.

ment 6A amounted to 5 grm., and in Experiment 6B to 10 grm.; notwithstanding this difference the Curve VIB lies above the Curve VIA. This experiment proves, therefore, again, that the muscle is deformed, more especially by strong stimuli (Table LIX, fig. 76).

Experiment 5 of Cybulski and Zanietowski yields the same result.

Finally, I reproduce an experiment where the authors produced alternately a twitch and a tetanus of short duration (Table LX, fig. 77). The discharge current of the condenser excited the nervus ischiadicus of a nerve-muscle preparation of the frog. The tetani were obtained by repeating the discharge at a rate of 22·5 per second. We see that in the case of tetani of short duration the formula is still applicable, but that the constants are different in the two instances.

Cybulski and Zanietowski also observed the phenomenon that the lift may decrease by increasing stimulus, when exciting the muscle directly, but they attributed it to fatigue (loc. cit., p. 142).

(IV) *Experiment of Waller* [45], 1895.—Waller used the nerve-muscle preparation of the frog. The nerve was excited by induction shocks of increasing strength for periods of one-eighth of a second. The interval between the successive stimuli is one minute. Table LXI contains the data of Waller's experiment, and fig. 78 shows how far the observed and calculated values of the lift agree.

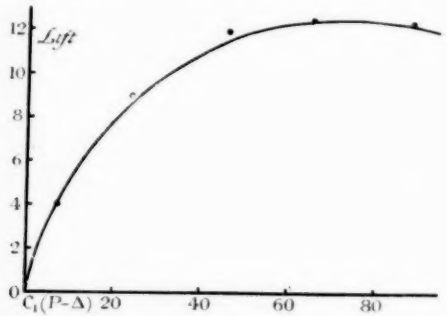

FIG. 78.—Graphic representation of an experiment of Waller. The lift as a function of the reduced stimuli.

(V) *Experiments of Keith Lucas* [30], 1909.—The author used the M. cutaneus dorsi of the frog. The animal was plunged in a bath of Ringer solution. The proprioceptive reflex arc was intact, so that he worked under the same experimental conditions as I did. The nerve on its course to the muscle was stimulated with a series of break-induction currents. The mean intensity of the current is by some indirect way determined by means of a ballistic galvanometer. The strength of the current increased in steps in the successive stimulations. These steps, however, were very small, so that often the passive resistances of the physiological system are not overcome. Hence, two or even three stimuli of increasing strength yield often the same lift.

Experiment 6 *of Keith Lucas* (Table LXII, fig. 79).—"In this experiment the exciting current was not graded finely during the first few observations, so that the steps of the rise are not seen." I choose this experiment exactly for that reason. The experiment shows clearly the deviation at the beginning, and also the existence of a maximum at P = 45·5. Beyond the top the plastic deformation by the supramaximal stimuli is obvious. Experiments 1 and 1A prove also the agreement between the observed and the calculated values of the lift. Especially for the larger values of P the agreement is evident.

(VI) *Experiments of Gildemeister* [20], 1911.—Gildemeister used the discharge current of a condenser, with which he stimulated a nerve-muscle preparation.

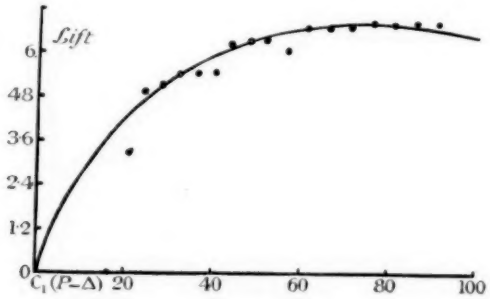

FIG. 79.—Graphic representation of Experiment 6 of Keith Lucas. The lift as a function of the reduced stimuli.

Experiment 11 *of Gildemeister* (Table LXIII, fig. 80).—A nerve-muscle preparation of the frog is used for this experiment. Every observation is repeated twice. The experiment is composed of two sets of observations. The observations of Curve I are obtained by means of quick discharges of the condenser, and those of Curve II are the result of slow discharges. Because the stimuli in the two cases are qualitatively different as the rate of discharge varies, the constants are different in the two sets of observations.

Experiment 5 *of Gildemeister* is of the same kind and yields similar results.

(VII) *Experiments of May* [32] 1911, 1912.—May worked with the tonic M. triceps suræ of the cat. The animal was slightly narcotized with chloroform. The muscle remained in normal relation with the nervous system, so that the proprioceptive reflex arc was intact. The Achilles

tendon was cut through, the muscle carefully loosened and attached to the rectilinear myograph of Keith Lucas. A normal salt solution at body temperature flowed through the muscle in order to eliminate the influence of the chloroform upon the muscle. May operated therefore with a normal tonic muscle and under the same conditions as I did. The muscle was stimulated either directly or indirectly through the motor nerve, by a current of 2 milliamperes, interrupted by a Leduc interrupter. The number of interruptions per second was chosen in such a way that each elementary current could display its full physio-

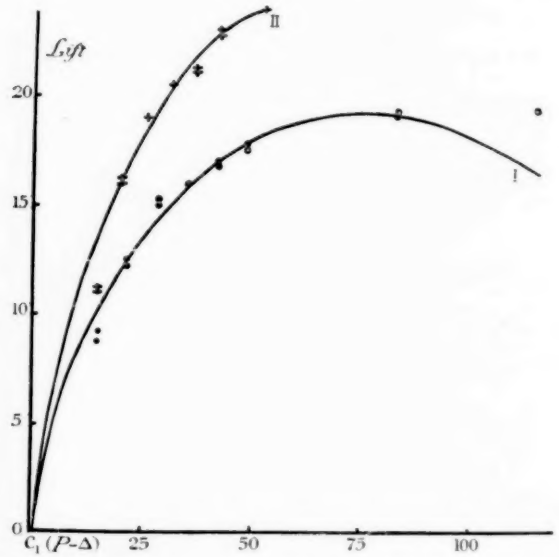

FIG. 80.—Graphic representation of Experiment 11 of Gildemeister. The lift as a function of the reduced stimuli.

logical effect, and that the strength of each elementary current amounted to but a fraction of a milliampere. The appliance of a Leduc interrupter makes it possible to work with currents of such a feeble intensity, and the increment of the stimulus is obtained by augmenting the frequency of the interruptions. The numbers in the second column of Tables LXIV, LXV, LXVI, denote the number of interruptions per second. The stimulation always lasted one second and the muscle was loaded with 50 or 100 grm. The use of very weak currents is probably the cause that plastic deformation seems to play no rôle in

these experiments. The absence of any perceptible deformation follows not only from my calculation, but the author himself remarks that "if the frequency instead of being increased is diminished step by step, the tracing obtained is the negative image of those obtained by increasing stimuli."

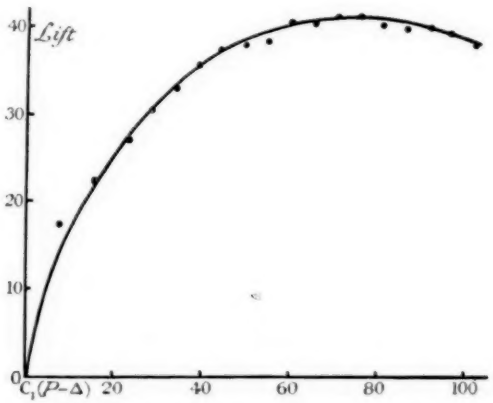

FIG. 81.—Graphic representation of Experiment 2 of May. The lift as a function of the reduced stimuli.

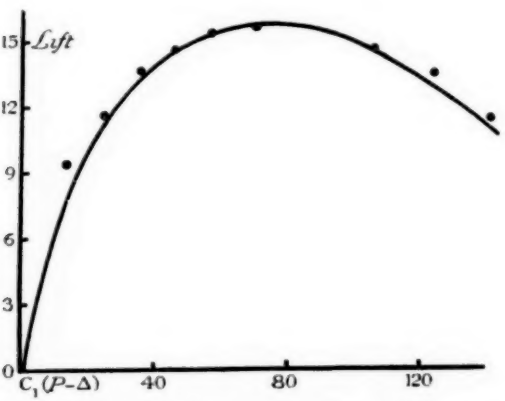

FIG. 82.—Graphic representation of Experiment 3 of May. The lift as a function of the reduced stimuli.

Experiment 2 of May (Table XLIV, fig. 81).—The muscle was excited directly by two platinum iridium electrodes, one placed under the top of the muscle, the other near the Achilles tendon. The curve

shows obviously a top in the neighbourhood of P = 150 and then decreases. In this case the agreement, especially for the decreasing part of the curve, is evident, because no deformation occurs.

Experiment 3 of May (Table LXV, fig. 82).—This experiment is of the same kind as the preceding one. In this case the decrease of the

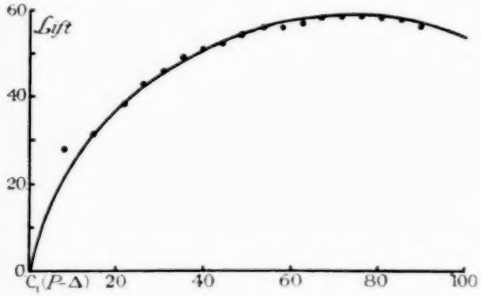

FIG. 83.—Graphic representation of Experiment 4 of May. The lift as a function of the reduced stimuli.

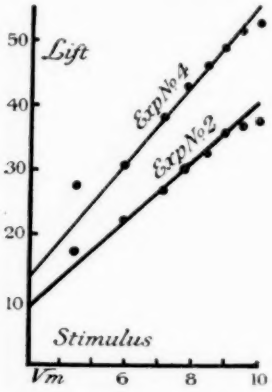

FIG. 84.—Graphic representation of Experiments 2 and 4 of May. The lift as a function of the square root of the number of interruptions (*m*).

lift by increasing stimulus is very considerable. This experiment is therefore remarkable, because it shows the close agreement between the observed and the calculated values of the lift for a considerable part of the descending branch of the curve. This fact is the more remarkable, because the curve is not symmetrical in respect to the maximum ordinate.

Experiment 6 is of the same nature and proves also the close agreement between the observed and the calculated values of the lift.

Experiment 4 *of May* (Table LXVI, fig. 83).—In this experiment the nervus ischiadicus was stimulated with very weak currents. As may be seen from fig. 83, the agreement between the observed and the calculated values of the lift are very close.

Finally, I thought it interesting to test the renowned formula of Nernst [34] on the experiments of May. The formula of Nernst applied in this case leads to the result that the increment of the lift ought to be proportionate to the square root of the number of interruptions. In fig. 84 I plotted these square roots against the lifts. We see, indeed, that the agreement is very close in the beginning, but that the divergence begins in the neighbourhood of the sixth observation. The physical conception of Nernst of the physiological problem is, therefore, confirmed by these experiments, at least for small values of P.

We conclude from all these experiments that the formula

$$-l = \frac{1}{C_1 C_2}\left[C_1(P-\Delta)\left\{\lg n.\, C_1(P-\Delta) - 1\right\}\right]$$

really represents the relation between stimulus and lift, inasmuch as a formula which contains only one independent variable may be able to represent this relation. Hence we conclude *that the analysis of the extension curve of the tonic muscle, which leads to the formula* $(-l)$, *is confirmed by the facts.*

More especially we may conclude, in agreement with Wedenski [46], Woolley [48], and others, *that the lift considered as a function of the stimulus in physical units is a maximum phenomenon, and that the lift decreases when the stimuli become supramaximal. This decrement of the lift is in most cases, however, counterbalanced, and therefore hidden, by the plastic deformation of the muscle.*

Chapter IV.—Tonus and Tendon Reflex.

The analysis of the extension curves of the tonic muscle has led to the result that the tonic muscle is continually in a state of slight contraction, combined with a state of exalted plasticity. The source of the stimuli which maintain this peculiar state of the muscle are the movements of the body which excite continuously the proprioceptors of the whole apparatus of locomotion. The physiological facts brought into connexion with the morphological discoveries of Boeke caused us to conclude that plasticity is the chief property of the sarcoplasmatic

part of the muscle and that the maintenance of a slight state of contraction is due to the striped apparatus. Hence one component of the tonus, the plasticity, is controlled by the sympathetic system, and the other component, the contraction, remains under the control of the motor cell of the anterior horn.

From the two components, by which muscle tonus is composed, the component "contraction" is the more variable. For, in the first place, preformed spinal mechanisms may influence the state of contraction of the muscle, and in the second place voluntary impulses may change the state of contraction. The plasticity, on the contrary, as being the autonomic component of the tonus, is withdrawn from our will, and therefore is the more stable component. The clinical definition, which is usually given of muscle tonus, viz., a state of slight contraction, keeps only one component in view, and especially the more variable one. Hence it is obvious that clinical observations, as well as experiments, based upon that definition have given contradictory results, and have failed to prove beyond a doubt the existence of tonus.

It seems desirable to me to have clinical terms to designate the two components of the tonus, and I should be inclined to propose the terms "*contractile tonus*" and *plastic*,[1] or *autonomic, tonus*, as perhaps the most adequate ones.[2] Instruments suitable for the measurement of the two different qualities of the tonus separately do not exist at the moment, as far as I know. Hence I will give a rather rough, but in many respects sufficient, clinical method. The M. biceps of the arm may for this purpose serve as an example. We begin to divert the attention of the patient from what we are doing, because attention markedly influences the state of contraction of our muscles. Next we take the wrist with one hand, fix the shoulder with the other and then we bend and extend the arm several times. Finally, we extend the arm moderately and take the belly of the biceps between thumb and fingers of the hand which fixed the shoulder. Now we first try to displace the muscle to and fro across its length. The resistance felt by this movement gives us an impression of the state of contraction of the muscle, or of the contractile tonus; next we compress the belly of the muscle gently

[1] Sherrington (*Quart. Journ. of Exp. Phys.*, 1909, vol. iii) uses also the term "plastic tonus," but in a somewhat different sense.

[2] The investigations on the tonus of smooth muscles have led to similar results. Plasticity, for instance, agrees in its essential features with "Substanztonus" of P. Schultz, and the "neurogene tonus" of the same author resembles in many respects contractile tonus. This agreement is not accidental, since the saroplasmatic part of the muscle is morphologically and physiologically the analogue of the smooth muscle.

between thumb and index and this compression affords us an impression of the plasticity of the muscle, or of the plastic tonus. If we repeat this proof two or three times, we succeed fairly well in getting an idea of the tonic state of the muscle. The repeated movement of bending and extending the arm serves to activate the proprioceptive reflex arc and to eliminate as much as possible the influence of accidental stimuli upon the tonus of the M. biceps. Because the two components of the tonus are independent of each other we may find little resistance by the displacement of the muscle combined with marked plasticity, or the reverse, but in most cases resistance and plasticity go *pari passu* under normal conditions.

I do not think that it will be easy to construct a simple instrument, suitable for clinical use to measure contractile tonus, but the degree of plastic tonus could perhaps be estimated by means of a set of test-cylinders of rubber of varying plasticity. If we alternately compress the muscle and the test-cylinders, it will be possible to find out the cylinder which approximately equals the muscle in plasticity.

The tendon phenomena have always been considered as connected with muscle tonus, but about the nature of this connexion opinions are divided. The majority of the observers believe that the tonic state of the muscle is a true reflex action, and the tendon phenomenon a direct muscular response, of which only the tonic muscle is capable (Gowers and others). The chief argument, upon which this supposition is based, is rather a negative one—namely, that the latent period between the tap on the tendon and the contraction is too short for a reflex. Of recent years, however, Sherrington [41], Snijder [42], and others, have found indubitable muscular reflexes with as short a latent period as the tendon phenomenon.

In my opinion, in agreement with the minority of the clinical observers (Snijder and others), *the tendon phenomenon is a true reflex, the typical reflex manifestation of the proprioceptive reflex arc.* The tap on the tendon is the stimulus which elicits reflexly the contraction. The knee-jerk may serve as example for the description of the mechanism of the tendon reflex. If we will elicit a knee-jerk, we begin to stretch the M. quadriceps by bending the knee sufficiently. The tap on the tendon produces under these circumstances a brief and sudden increment of the stress of the muscle. The duration of this variation of the stress, however, is too short to produce a displacement or a lengthening of the muscle as a whole, but it evokes a stress-wave, travelling through the muscle. This wave, which is directly visible,

may also be recorded by means of a tambour of Marey. The variation of the tension, provoked by the stress-wave, forms the adequate stimulus for the proprioceptors of the muscle and the tendon. The initial stretching of the muscle, is, therefore, necessary to create the physical condition needed for the production and the propagation of the stress-wave through the muscle. The response of the proprioceptive reflex arc to the stimulation of its proprioceptors is a contraction. The direct observation of the contraction as well as the records on a drum prove that one of the components of the contraction is a single twitch. The electric phenomena, accompanying the contraction, recorded by Wertheim Salomonson [47], and by Dittler and Günther [17], by means of the string galvanometer, are in full harmony with this. A close observation shows, however, that the stadium decrementi of the twitch always seems lengthened. This lengthening is very obvious in the tracings of Gotch [21]. Sometimes the descending branch of the twitch may exhibit a slow secondary elevation (Shepherd Franz [19]). The lengthening or the secondary elevation is caused by a tonic contraction. We conclude from these facts *that the contraction following the tap on the tendon is of a twofold nature, viz., a twitch upon which is superposed a tonic contraction.* That we have really to deal with a superposition of two different forms of contraction is convincingly proved by Graham Brown [22], de Boer [9], and others.

The two components composing the muscular contraction which follows reflexly the tap on the tendon, seem to be independent of each other. This is demonstrated also by an experiment of de Boer on cats. de Boer cut through the rami communicantes of the sympathetic chain, and destroyed in that way the plastic tonus. Notwithstanding the loss of the plastic tonus, the tendon reflex could be elicited and was even brisk. This briskness of the tendon reflex is probably caused by the absence of the tonic contraction. Clinical observation tends to the same conclusion. There are, namely, some forms of locomotor ataxia in which the twitch is diminished or absent, and in these cases the slow and sluggish contraction seems caused only by the tonic component. Also a partial dissociation between the twitch and the tonic contraction occurs.

Shortly summarizing, we may say that the term " tonus " designates the state of the muscle when continually subjected to the influence of weak stimuli, emanating from its own proprioceptors. By the term " tendon reflex " we indicate the sudden variation of the tonus, evoked reflexly by a brief but vigorous stimulus of the proprioceptors of the

muscle and the tendon. Hence *tonus and tendon reflex as to their intimate nature are identical*, and the tendon reflex may therefore serve as a touchstone for the tonic state of the muscle. *In the tonus however, the plastic component seems to prevail, and in the tendon reflex the twitch is the most obvious phenomenon. Hence it is evident that the tendon reflex will afford us especially an impression about the contractile component of the tonus.*

It follows from our conception that the tendon phenomenon is a true reflex, and that the muscular response is of a twofold nature, that the terms used to designate the different modalities of the tendon reflex are very imperfect. I will leave aside here the question of the relation between the intensity of the tap on the tendon and the duration of the latent period between the tap and the contraction,

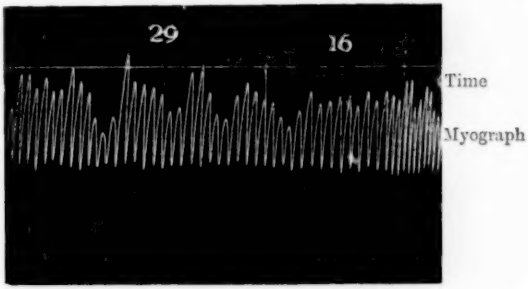

FIG. 85.—Clonus tracing of the tonic M. triceps suræ of the spinal cat. The speed of the drum was not uniform, as may be seen at the time marking.

as well as the question of the threshold value. What interests us, clinically, is the relation between the intensity of the tap on the tendon and the mechanical effect. This effect may be normal, diminished, or exaggerated, but these terms remain equivocal unless we add, which of the two components is normal, diminished, or exaggerated. Under normal conditions the variation of the two components usually goes *pari passu*, but under pathological conditions this is no more the case. When the twitch prevails, we get the impression that the reflex is brisk, and when the tonic contraction dominates that it is slow. I think, therefore, that the combination of the terms "normal, diminished, exaggerated," with brisk or slow will be sufficient for practical use, though they do not cover the whole field of phenomena.

When the irritability of the reflex centre is exalted, and the tendon reflex exaggerated, the stimulation of the proprioceptors, called forth by the reflex contraction, suffices to again engender a contraction. This repetition of the reflex is called "clonus."

Fig. 85 is the reproduction of a clonus tracing of the M. triceps suræ of a spinal cat. It is evident from this tracing that clonus is a series of pure twitches, in which no trace of a tonic contraction is to be detected.[1] This is always the case in clonus tracings in man. Fig. 86 represents another clonus curve, also of a spinal cat. At the moment indicated by the dropping of the signal line, the load attached on the

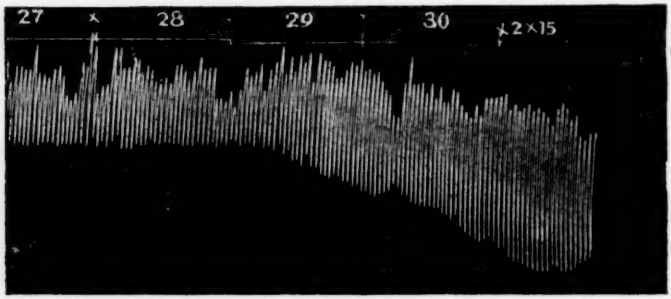

FIG. 86.—Clonus tracing of the tonic M. triceps suræ of the spinal cat. At the moment indicated by the dropping of the signal line, the load begins to increase uniformly.

tendon of the muscle is uniformly increased. The increasing load acting as a stimulus for the proprioceptors, accelerates the rhythm of the clonus by about 7 per cent., and approximately doubles the excursion of the movement.

Figs. 87 and 88 show what precedes the commencement of the clonus. At A the muscle exhibits a tonic shortening, then the length of the muscle remains constant till B. Again the muscle shortens, but this shortening is accompanied by a clonus. In the case represented by fig. 88 the clonus was initiated by a jerk at the tendon [×]. A clonus, like a tendon-reflex, is in most cases initiated by a jerk or a pull at the tendon, but sometimes (as in fig. 87) the clonus seems pseudo-spontaneous. In these last cases there are usually movements in antagonistic groups of muscles which initiate the clonus of the agonist. It follows from my experiments that the beginning of a clonus is always accom-

[1] This fact agrees with the form of the action current as recorded by Wertheim Salomonson [47].

panied by a tonic shortening, but that inversely a tonic shortening is not always followed by a clonus. Hence I conclude that the tonic shortening is a necessary factor in the production of a clonus, or *that a clonus is composed by a series of twitches superposed upon a tonic shortening.*

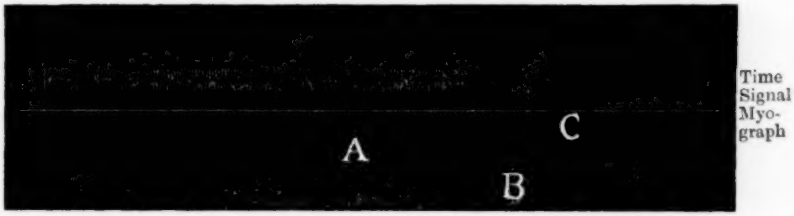

FIG. 87.—Clonus tracing of the tonic M. triceps suræ of the spinal cat. At A and at B a tonic shortening preceding the clonus; at C tonic lengthening of the muscle following the cessation of the clonus.

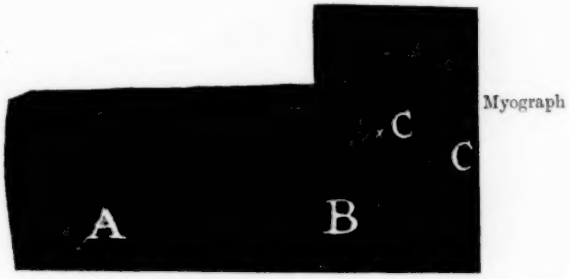

FIG. 88.—Clonus tracing of the tonic M. triceps suræ of the spinal cat. At A and at B a tonic shortening preceding the clonus; at C tonic lengthening of the muscle following the cessation of the clonus; at × the clonus is initiated by a jerk at the tendon.

The cessation of a clonus is always accompanied by a tonic lengthening of the muscle (figs. 87, 88, C). Fig. 89, also a clonus from the triceps suræ of a spinal cat, shows a peculiarity of the tonic lengthening. At the beginning of the experiment, the muscle slowly lengthens, then, at the moment indicated by a cross, I pulled at the tendon, and a clonus set in. Very soon the clonus dies away, and at C the tonic lengthening begins. Now the line representing the tonic lengthening shows clearly the staircase phenomenon. This staircase phenomenon is also visible upon the line of the tonic shortening. This line, however, is in most cases very steep, and this is the reason

that the steps are not very obvious. The ascending line of fig. 89 exhibits two steps, each a little below the top of the first and the second twitch. We conclude from these facts *that the tonic shortening of the muscle preceding the clonus is a tonic tetanus, and that this tetanus is the result of the exalted irritability of the proprioceptive reflex arc. A clonus is therefore a series of twitches elicited reflexly, and superposed upon a tonic tetanus.* This result is in harmony with our conception of the duality of the tendon reflex.

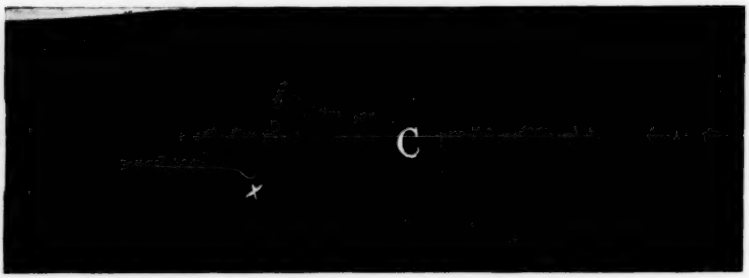

FIG. 89.—Clonus tracing of the tonic M. triceps suræ of the spinal cat. At [×] the clonus is initiated by a pull at the tendon. At C a slow tonic lengthening of the muscle. The line representing the lengthening of the muscle shows the staircase phenomenon.

We arrive, therefore, at the general result that the tonus, the tendon reflex, and the clonus are closely allied phenomena, and that they are composed by an element "contraction," due to the action of the motor cell of the anterior horn and by an element "plasticity," due to the action of a sympathetic motor cell of the cord. *In the tonus the autonomic component prevails; in the tendon reflex the twitch dominates the tonic contraction; in the clonus a series of twitches is superposed upon a tonic tetanus.*

The results at which we have arrived, viz., the morphological and functional duality of the striped muscle, may throw light upon several problems of pharmacology and neuropathology.

My experiments tend, for instance, to the conclusion that several drugs, e.g., strychnine, affect preponderantly the sarcoplasmatic part of the muscle. Fig. 90 is a reproduction of a reflex contraction of the M. gastrocnemius of the frog, elicited by stimulation of the skin of the foot. The twitch does not seem obviously altered, but the height and the duration of the tonic contraction (tC) are strongly increased. The same effect may be produced by cooling down the whole animal.

The cutting through of the brain-stem between thalamus and mid-brain (Sherrington [40]) provokes a similar effect. By this section the lower sympathetic centres are divided from the higher centres, situated in the base of the brain. I have only qualitative experience of the state, designated by Sherrington "decerebrate rigidity"; but it seems very probable to me that this rigidity is at least partly due to a spasm of the sarcoplasmatic part of the muscle.[1] Hence one of the components in the decerebrate rigidity is a spasm of sympathetic origin, caused by the prevailing of the cerebellum. This opinion harmonizes

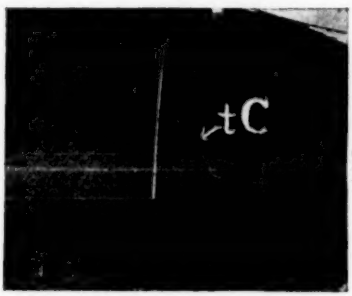

FIG. 90.—Reflex contraction of the tonic M. gastrocnemius of the frog, elicited by stimulation of the skin of the foot, after injection of strychnine. (tC) tonic part of the contraction. The enlargement is about 10 times. The time is marked in tenths of seconds.

with the views of Sherrington [41], who considers the cerebellum as the main ganglion of the proprioceptive system.

Another example produces the myopathic forms of progressive muscular atrophy. The muscle by its sympathetic innervation comes upon the same line with the other internal organs of the body, which all belong to the domain of the sympathetic system. The pathological anatomy of these myopathies make it probable that not only the muscle is diseased, but also a part of the sympathetic system [31]. The primary motor neurone, on the contrary, is always found normal—at least in the beginning of the disease. In this respect the myopathic forms differ from the neurotic forms of muscular atrophy, in which the primary motor neuron is affected.

These few examples may suffice to show that the conception of the duality of the striped muscle may be very fruitful in the future for our knowledge of the nervous system.

[1] The experiments of Dusser de Barenne tend to the same conclusion (*Folia Neurob.*, 1913, Bd. vii, B. 651).

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APPENDIX.

[THESE TABLES SHOULD BE READ IN CONJUNCTION WITH CHAPTER I.]

(*A*) *The Load Variable.*

TABLE I.—*Experiment* 1 (November 9, 1904).

τ	L	$\frac{\Delta L}{\Delta \tau}$	$\frac{\Delta L}{\Delta \tau} \times \tau$
0·0 × 60	0·0	—	—
0 + ϵ	109·0 (?)	—	—
$\frac{1}{16}$	—	64·0	4·0
$\frac{1}{8}$	117·0	—	—
$\frac{3}{16}$	—	20·0	3·75
$\frac{1}{4}$	119·5	—	—
$\frac{3}{8}$	—	11·0	4·12
$\frac{1}{2}$	122·5	—	—
$\frac{3}{4}$	—	5·0	3·75
1	124·75	—	—
$1\frac{1}{4}$	—	2·75	4·12
2	127·5	—	—
$2\frac{1}{2}$	—	1·5	3·75
3	129·0	—	—
4	—	1·0	4·0
5	131·0	—	—
6	—	0·625	3·75
7	132·25	—	—
$8\frac{1}{2}$	—	0·50	4·25
10	133·75	—	—
$12\frac{3}{4}$	—	0·32	4·0
$15\frac{1}{2}$	135·5	—	—
18	—	0·20	3·6
$20\frac{1}{2}$	136·5	—	—
23	—	0·20	4·6
$25\frac{1}{2}$	137·5	—	—
28	—	0·15	4·2
$30\frac{1}{2}$	138·25	—	—
$35\frac{1}{2}$	—	0·125	4·4
$40\frac{1}{2}$	139·5	—	—
46	—	0·09	4·0
$51\frac{1}{2}$	140·5	—	—
$51\frac{3}{4}$ × 60	—	—	—
			a.m. 4·02
0 = $51\frac{3}{4}$ × 60	140·5	—	—
$51\frac{3}{4}$ + ϵ	82·25 (?)	—	—
$51\frac{3}{16}$	—	56·0	3·5
$51\frac{1}{4}$	75·25	—	—
$51\frac{1}{16}$	—	18·0	3·4
52	73·0	—	—
$52\frac{1}{2}$	—	10·0	3·75
$52\frac{1}{4}$	70·5	—	—

TABLE I.—*continued.*

τ	L	$\frac{\Delta L}{\Delta \tau}$	$\frac{\Delta L}{\Delta \tau} \times \tau$
$52\frac{3}{4}$	—	5·0	3·12
$52\frac{1}{2}$	69·25	—	—
$52\frac{3}{4}$	—	3·5	3·5
53	67·5	—	—
$53\frac{1}{2}$	—	2·0	3·5
54	65·5	—	^^^^^
$54\frac{1}{2}$	—	0·75 (?)	—
55	64·75	—	—
$55\frac{1}{2}$	—	1·0	3·75
56	63·75	—	—
$56\frac{1}{4}$	—	0·71	3·6
$57\frac{3}{4}$	62·5	—	—
$59\frac{3}{4}$	—	0·44	3·5
$61\frac{3}{4}$	60·75	—	—
$64\frac{1}{4}$	—	0·25	3·12
$66\frac{3}{4}$	59·5	—	—
$69\frac{1}{4}$	—	0·2	3·5
$71\frac{3}{4}$	58·5	—	—
$76\frac{3}{4}$	—	0·15	3·75
$81\frac{3}{4}$	57·0	—	—
$86\frac{3}{4}$	—	0·1	3·5
$91\frac{3}{4}$	56·0	—	—
$94\frac{3}{4}$	—	—	—
$96\frac{3}{4} \times 60$	55·5	$<0·1$	$<4·25$
		$>0·05$	$>2·1$
			a.m. 3·5

TABLE II.—*Experiment* 2 (November 7, 1904).

τ	L	$\frac{\Delta L}{\Delta \tau}$	$\frac{\Delta L}{\Delta \tau} \times \tau$
$0·0 \times 60$	0·0	—	—
$0 + \epsilon$	121·5 (?)	—	—
$\frac{1}{16}$	—	96·0	6·0
$\frac{1}{8}$	133·5	—	—
$\frac{3}{16}$	—	32·0	6·0
$\frac{1}{4}$	137·5	—	—
$\frac{1}{2}$	—	16·0	6·0
$\frac{1}{2}$	141·5	—	—
$\frac{3}{4}$	—	8·0	6·0
1	145·5	—	—
$1\frac{1}{2}$	—	4·0	6·0
2	149·5	—	—
$2\frac{1}{2}$	—	2·5	6·25
3	152·0	—	—
4	—	1·5	6·0
5	155·0	—	—
$7\frac{1}{2}$	—	0·8	6·0
10	159·0	—	—
$12\frac{1}{2}$	—	0·5	6·25
15	161·5	—	—
$17\frac{1}{2}$	—	0·35	6·12
20	163·0	—	—
$22\frac{1}{2}$	—	0·3	6·75

TABLE II.—*continued.*

τ	L	$\frac{\Delta L}{\Delta \tau}$	$\frac{\Delta L}{\Delta \tau} \times \tau$
25	164·5	—	—
27½	—	0·25	6·85
30	166·0	—	—
32½	—	0·2	6·5
35	167·0	—	^^^^^
37½	—	0·25 (?)	—
40	168·25	—	—
42½	—	0·1 (?)	—
45	168·75	—	—
47½	—	0·15	6·6
50	169·5	—	^^^^^
55	—	0·15 (?)	—
60	171·0	—	—
65½	—	0·09	6·0
71 × 60	172·0	—	—
			a.m. **6·22**
0 = 71 × 60	172·0	—	—
71 + ϵ	104·5 (?)	—	—
71$\frac{1}{10}$	—	128·0	8·0
71$\frac{2}{10}$	88·5	—	—
71$\frac{3}{10}$	—	40·0	7·5
71$\frac{4}{10}$	83·5	—	—
71$\frac{5}{10}$	—	21·0	7·9
71$\frac{6}{10}$	78·25	—	—
71$\frac{7}{10}$	—	13·0	8·1
71$\frac{8}{10}$	75·0	—	—
71$\frac{9}{10}$	—	10·0	8·7
72	72·5	—	—
72$\frac{1}{2}$	—	5·0	7·5
72$\frac{2}{2}$	69·0	—	—
72$\frac{3}{2}$	—	4·0	7·0
73	67·0	—	—
73$\frac{1}{2}$	—	3·5	8·7
74	63·5	—	—
74$\frac{1}{2}$	—	2·5	8·7
75	61·0	—	—
75$\frac{1}{2}$	—	1·5	7·0
76	59·5	—	—
77	—	1·25	7·5
78	57·0	—	—
79	—	1·0	8·0
80	55·0	—	—
82$\frac{1}{2}$	—	0·7	8·0
85	51·5	—	—
87$\frac{1}{2}$	—	0·5	8·2
90	49·0	—	—
100	43·7	—	—
200	34·0	—	—
300	30·0	—	—
400	27·3	—	—
Extrapolation			
			a.m. **7·91**

TABLE III.—*Experiment* 3 (November 15, 1904).

τ	L	$\frac{\Delta L}{\Delta \tau}$	$\frac{\Delta L}{\Delta \tau} \times \tau$
00·0 60	0·0	—	—
0 + ε	108·5 (?)		
	—	36·0	4·5
	117·5		
	—	12·0	4·5
	120·5		
	—	7·0	5·0
1	123·5		
1½	—	3·0	4·5
2	126·5		
3	—	1·5	4·5
4	129·5		
6	—	0·75	4·5
8	132·5		
12	—	0·375	4·5
16	135·5		
24	—	0·19	4·5
32	138·5		
40	—	0·125	5·0
48	140·5		^^^^^
56	—	0·125 (?)	—
64	142·5		
72	—	0·063	4·5
80	143·5		
88	—	0·05	4·9
96	144·5	—	—
101 60	144·5	—	—
			a.m. 4·63
0 − 101 × 60	144·5	—	—
101 + ε	71·0 (?)		
101½	—	28·0	3·5
101½	64·0		
101⅔	—	9·0	3·4
101⅚	62·0		
101¾	—	4·0	3·0
102	60·0		
102½	—	2·0	3·0
103	58·0		
105	—	0·94	3·75
107	54·25		
110	—	0·42	3·75
113	51·75	—	—
117	—	0·22	3·5
121	50·0		
125	—	0·125	3·0
129	49·0	—	—
			a.m. 3·36

TABLE IV.—*Experiment* 4 (November 15, 1904).

τ	P	Δ P per 60 sec.	L	τ	P	Δ P per 60 sec.	L
0·0 × 60 ..	13·00		0·0	45·0 ..	42·01		59·0
3·0 ..	19·89	+ 2·30	14·6	48·0 ..	48·02	+ 2·00	61·75
6·0 ..	26·78		29·5	51·0 ..	54·03		65·5
7·5 ..	23·50	− 2·19	28·5	52·5 ..	50·31	− 2·48	65·0
9·0 ..	20·22		26·0	54·0 ..	46·58		64·0
12·0 ..	26·85	+ 2·21	34·0	57·0 ..	52·54	+ 1·99	66·0
15·0 ..	33·48		42·0	60·0 ..	58·50		70·0
16·5 ..	30·01	− 2·32	41·0	61·5 ..	54·77	− 2·49	69·5
18·0 ..	26·52		39·5	63·0 ..	51·04		68·0
21·0 ..	32·89	+ 2·12	44·0	66·0 ..	57·00	+ 1·99	70·5
24·0 ..	39·26		49·5	69·0 ..	62·97		74·0
25·5 ..	35·06	− 2·40	49·0	70·5 ..	59·24	− 2·49	73·5
27·0 ..	32·06		47·0	72·0 ..	55·50		72·0
30·0 ..	38·27	+ 2·07	50·5	75·0 ..	61·44	+ 1·98	74·0
33·0 ..	44·49		55·5	78·0 ..	67·39		77·5
34·5 ..	40·85	− 2·43	55·0	79·5 ..	63·66	− 2·49	77·0
36·0 ..	37·21		53·0	81·0 ..	59·92		76·0
39·0 ..	43·32	+ 2·04	56·5	84·0 ..	67·42	+ 1·98	78·75
42·0 ..	49·43		61·5	87·0 × 60 ..	71·81		81·0
43·5 ..	45·72	− 2·47	61·0				

TABLE V.—*Experiment* 5 (November 16, 1904).

τ	P	Δ P per 60 sec.	L	τ	P	Δ P per 60 sec.	L
0·0 × 60 ..	13·45		0·0	64·5 ..	42·32	− 2·26	67·0
1·5 ..	18·02	+ 3·05	8·5	66·0 ..	38·93		65·75
3·0 ..	22·06		19·0	67·5 ..	43·28	+ 2·90	67·5
4·5 ..	19·42	− 2·12	16·0	69·0 ..	47·64		71·0
6·0 ..	16·23		11·0	70·5 ..	44·24	− 2·27	70·25
7·5 ..	20·79	+ 3·01	18·0	72·0 ..	40·84		68·75
9·0 ..	25·35		26·0	73·5 ..	45·18	+ 2·89	71·0
10·5 ..	22·15	− 2·13	24·5	75·0 ..	49·52		74·0
12·0 ..	18·95		20·75	76·5 ..	46·10	− 2·28	73·5
13·5 ..	23·48	+ 3·02	26·0	78·0 ..	42·68		72·0
15·0 ..	28·02		33·0	79·5 ..	47·00	+ 2·88	74·0
16·5 ..	24·79	− 2·16	31·0	81·0 ..	51·33		77·5
18·0 ..	21·55		27·0	82·5 ..	47·89	− 2·29	76·25
19·5 ..	26·05	+ 3·00	32·0	84·0 ..	44·45		75·0
21·0 ..	30·55		39·0	85·5 ..	48·76	+ 2·87	76·5
22·5 ..	27·29	− 2·18	36·75	87·0 ..	53·06		79·0
24·0 ..	24·02		33·0	88·5 ..	49·59	− 2·32	78·25
25·5 ..	28·49	+ 2·98	38·0	90·0 ..	46·11		77·25
27·0 ..	32·97		44·0	91·5 ..	50·42	+ 2·87	78·5
28·5 ..	29·69	− 2·19	42·5	93·0 ..	54·72		81·0
30·0 ..	26·40		39·0	94·5 ..	51·28	− 2·29	80·5
31·5 ..	30·85	+ 2·97	43·0	96·0 ..	47·84		79·75
33·0 ..	35·31		49·5	97·5 ..	52·17	+ 2·89	81·0
34·5 ..	32·00	− 2·21	48·0	99·0 ..	56 50		83·25
36·0 ..	28·68		45·0	100·5 ..	53 10	− 2·27	83·0
37·5 ..	33·10	+ 2·95	48·5	102·0 ..	49·70		82·25
39·0 ..	37·53		53·5	103·5 ..	54·07	+ 2·91	83·5
40·5 ..	34·20	− 2·22	52 2	105·0 ..	58·44		86·0
42·0 ..	30·86		50·0	106·5 ..	55·07	− 2·25	85·5
43·5 ..	35·27	+ 2·94	53·0	108·0 ..	51·69		85·0
45·0 ..	39·67		57·0	109·5 ..	56·09	+ 2·93	86·0
46·5 ..	36·32	− 2·24	56·5	111·0 ..	60·49		89·5
48·0 ..	32·96		54·5	112·5 ..	57·19	− 2·20	89·25
49·5 ..	37·35	+ 2·93	57·0	114·0 ..	53·89		88·0
51·0 ..	41·74		60·75	115·5 ..	58·39	+ 3·00	90·0
52·5 ..	38·37	− 2·25	60·0	117·0 ..	62·89		92·5
54·0 ..	35·00		58·75	118·5 ..	59·67	− 2·15	92·25
55·5 ..	39·37	+ 2·92	61·0	120·0 ..	56·44		91·25
57·0 ..	43·75		64·25	121·5 ..	61·04	+ 3·07	93·0
58·5 ..	40·37	− 2·25	63·5	123·0 ..	65·64		96·0
60·0 ..	36·99		62·0				
61·5 ..	41·35	+ 2·91	64·5				
63·0 ..	45·71		67·75				

TABLE VI.—*Experiment* 6 (December 30, 1904).

P		τ		L		$\frac{\Delta L}{\Delta \tau}$		$\frac{\Delta L}{\Delta \tau} \times \tau$
4·8	..	0·0 × 60	..	0·0	..	—	..	—
7·8	..	1	..	—	..	—	..	—
14·8	..	2	..	—	..	—	..	—
24·8	..	3	..	—	..	—	..	—
44·8	..	4	..	—	..	—	..	—
—	..	5	..	116·0	..	—	..	—
74·8	..	$0 = 5\frac{1}{10}$	..	132·0 (?)	..	—	..	—
—	..	$5\frac{1}{5}$	..	—	..	21·5	..	7·0
—	..	$5\frac{1}{4}$	..	146·0	..	—	..	—
—	..	6	..	—	..	8·0	..	7·2
—	..	$6\frac{1}{4}$	..	150·0	..	—	..	—
—	..	$6\frac{1}{2}$	..	—	..	4·25	..	7·0
—	..	$7\frac{1}{4}$	..	154·25	..	—	..	—
—	..	$7\frac{1}{2}$	..	—	..	2·75	..	7·3
—	..	$8\frac{1}{4}$	..	157·0	..	—	..	—
—	..	$8\frac{1}{2}$	..	—	..	2·0	..	7·0
—	..	$9\frac{1}{2}$	..	159·5	..	—	..	—
—	..	$10\frac{1}{2}$	..	—	..	1·37	..	7·1
—	..	$11\frac{1}{2}$	..	162·25	..	—	..	—
—	..	13	..	—	..	0·91	..	7·3
—	..	$14\frac{1}{2}$	..	165·0	..	—	..	—
—	..	$16\frac{1}{4}$	..	—	..	0·64	..	7·2
—	..	18	..	167·25	..	—	..	—
—	..	20	..	—	..	0·5	..	7·5
—	..	22	..	169·25	..	—	..	—
—	..	$23\frac{1}{2}$	..	—	..	0·41	..	7·5
—	..	25	..	170·5	..	—	..	—
—	..	$27\frac{1}{2}$	..	—	..	$\begin{cases} <0·35 \\ >0·30 \end{cases}$	..	$\begin{cases} <7·9 \\ >6·7 \end{cases}$
—	..	30	..	172·0	..	—	..	—
—	..	$32\frac{1}{2}$	..	—	..	$\begin{cases} <0·30 \\ >0·25 \end{cases}$	..	$\begin{cases} <8·2 \\ >6·9 \end{cases}$
—	..	35	..	173·5	..	—	..	—
—	..	$37\frac{1}{2}$	..	—	..	$\begin{cases} <0·25 \\ >0·20 \end{cases}$	..	$\begin{cases} <8·1 \\ >6·5 \end{cases}$
—	..	40	..	174·5	..	—	..	—
—	..	45	..	—	..	0·175	..	7·0
—	..	50	..	176·25	..	—	..	—
—	..	55	..	—	..	0·15	..	7·5
—	..	60	..	177·75	..	—	..	—
—	..	61	..	177·75	..	—	..	—
							a.m.	7·27
44·8	..	$0 = 61\frac{1}{10}$	..	166·75 (?)	..	—	..	—
—	..	$61\frac{3}{10}$	..	—	..	3·75	..	0·375
—	..	$61\frac{1}{2}$	..	166·0	..	—	..	—
—	..	$61\frac{1}{4}$	..	—	..	1·0	..	0·45
—	..	$61\frac{3}{4}$	..	165·5	..	—	..	—
—	..	$62\frac{1}{2}$	..	—	..	0·2	..	0·33
—	..	63	..	165·25	..	—	..	—
—	..	64	..	—	..	0·125	..	0·375
—	..	65	..	165·0	..	—	..	—
—	..	$67\frac{1}{2}$	..	—	..	0·05	..	0·33
—	..	70	..	164·75	..	—	..	—
—	..	75	..	—	..	0·025	..	0·35
—	..	80	..	164·5	..	—	..	—
—	..	85	..	—	..	$\begin{cases} <0·025 \\ >0·0125 \end{cases}$	..	$\begin{cases} <0·6 \\ >0·3 \end{cases}$
—	..	90	..	164·25	..	—	..	—
—	..	$91\frac{3}{4}$	..	164·25	..	—	..	—
							a.m.	0·39

TABLE VI.—*continued.*

	τ	L	Δ L / Δ τ	Δ L / Δ τ × τ
24·8 ..	0 = 91$\frac{10}{16}$ ..	153·75 (?) ..	— ..	—
— ..	91$\frac{10}{16}$ ..	— ..	3·0 ..	0·75
— ..	92 ..	152·25 ..	— ..	—
— ..	92$\frac{8}{16}$ ..	— ..	1·66 ..	0·71
— ..	92$\frac{12}{16}$ ..	151·0 ..	— ..	—
— ..	93$\frac{4}{16}$ ..	— ..	0·5 ..	0·77
— ..	94$\frac{4}{16}$ ..	150·25 ..	— ..	—
— ..	97$\frac{4}{16}$ ..	— ..	0·13 ..	0·67
— ..	100 ..	149·5 ..	— ..	—
— ..	105 ..	— ..	0·05 ..	0·65
— ..	110 ..	149·0 ..	— ..	—
— ..	115 ..	— ..	$\left\{\begin{array}{l}<0·036\\>0·025\end{array}\right.$ ..	$\left\{\begin{array}{l}<0·86\\>0·58\end{array}\right.$
— ..	120 ..	148·75 ..	— ..	—
— ..	121$\frac{7}{16}$ ..	148·75 ..	— ..	—
				a.m. **0·71**
14·8 ..	0 = 121$\frac{5}{16}$ ..	135·0 (?) ..	— ..	—
— ..	121$\frac{13}{16}$ ..	— ..	20·0 ..	1·0
— ..	122 ..	137·0 ..	— ..	—
— ..	122$\frac{1}{16}$ ..	— ..	4 ..	0·9
— ..	122$\frac{4}{16}$ ..	136·0 ..	— ..	—
— ..	122$\frac{9}{16}$ ..	— ..	1·5 ..	0·9
— ..	122$\frac{12}{16}$ ..	135·25 ..	— ..	—
— ..	123$\frac{8}{16}$ ..	— ..	0·66 ..	1·0
— ..	123$\frac{8}{16}$ ..	134·75 ..	— ..	—
— ..	124$\frac{8}{16}$ ..	— ..	0·37 ..	0·98
— ..	125$\frac{8}{16}$ ..	134·0 ..	— ..	—
— ..	127$\frac{8}{16}$ ..	— ..	0·17 ..	0·98
— ..	130 ..	133·25 ..	— ..	—
— ..	135 ..	— ..	0·075 ..	0·97
— ..	140 ..	132·5 ..	— ..	—
— ..	146 ..	— ..	0·042 ..	1·0
— ..	152 ..	132·0 ..	— ..	—
				a.m. **0·97**
7·8 ..	0 = 152$\frac{1}{16}$ ..	120·75 (?) ..	— ..	—
— ..	152$\frac{2}{16}$ ..	— ..	13·0 ..	1·62
— ..	152$\frac{8}{16}$ ..	117·5 ..	— ..	—
— ..	152$\frac{9}{16}$ ..	— ..	3·0 ..	1·5
— ..	153 ..	116·0 ..	— ..	—
— ..	153$\frac{8}{16}$ ..	— ..	1·25 ..	1·56
— ..	154 ..	114·75 ..	— ..	—
— ..	157 ..	— ..	0·33 ..	1·58
— ..	160 ..	112·75 ..	— ..	—
— ..	165 ..	— ..	0·125 ..	1·56
— ..	170 ..	111·5 ..	— ..	—
— ..	175 ..	— ..	0·075 ..	1·7
— ..	180 ..	110·75 ..	— ..	—
— ..	181 ..	110·75 ..	— ..	—
				a.m. **1·59**
4·8 ..	0 = 181$\frac{4}{16}$ ..	103·0 ..	— ..	—
— ..	181$\frac{8}{16}$ ..	— ..	10·0 ..	1·25
— ..	181$\frac{12}{16}$ ..	100·5 ..	— ..	—
— ..	182 ..	— ..	2·5 ..	—
— ..	182$\frac{4}{16}$ ..	99·25 ..	— ..	1·25
— ..	182$\frac{8}{16}$ ..	— ..	1·0 ..	—
— ..	183$\frac{4}{16}$ ..	98·25 ..	— ..	1·25
— ..	184$\frac{4}{16}$ ..	— ..	0·5 ..	—
— ..	185$\frac{4}{16}$ × 60 ..	97·25 ..	— ..	1·12
				a.m. **1·22**

TABLE VII.—*Experiment* 6 (December 30, 1904).

P	Δ P	$\frac{\Delta L}{\Delta \tau} \times \tau$	$\frac{\Delta L}{\Delta \tau} \times \frac{\tau}{\Delta P}$
74·8	—	—	—
—	30·0	—	—
44·8	—	0·39	0·013
—	20·0	—	—
24·8	—	0·71	0·036
—	10·0	—	—
14·8	—	0·97	0·097
—	7·0	—	—
7·8	—	1·59	0·230
—	3·0	—	—
4·8	—	1·22	0·41

TABLE VIII.—*Experiment* 7 (January 5, 1905).

P	τ	L	$\frac{\Delta L}{\Delta \tau}$	$\frac{\Delta L}{\Delta \tau} \times \tau$
4·8	0·0 × 60	0·0	—	—
7·8	0·0 + ε	14·0 (?)	—	—
—	$\frac{1}{4}$	—	12·0	1·5
—	$\frac{1}{2}$	17·0	—	—
—	$\frac{5}{8}$	—	3·0	1·5
—	$\frac{3}{4}$	18·5	—	—
—	1	—	1·5	1·5
—	$1\frac{1}{4}$	19·25	—	—
—	2	—	0·83	1·66
—	$2\frac{1}{4}$	20·5	—	—
—	$3\frac{1}{4}$	—	0·5	1·62
—	$3\frac{3}{4}$	20·75	—	—
—	$4\frac{3}{4}$	—	< 0·37	< 1·78
—			> 0·25	> 1·2
—	$5\frac{3}{4}$	21·5	—	—
—	$7\frac{1}{4}$	—	< 0·25	< 1·8
			> 0·17	> 1·2
—	$8\frac{3}{4}$	22·0	—	—
—	$11\frac{3}{4}$	—	< 0·17	< 1·96
			> 0·12	> 1·47
—	$14\frac{3}{4}$	23·0	—	—
				a.m. 1·56
14·8	0 = $15\frac{1}{4}$	41·5 (?)	—	—
—	$15\frac{3}{4}$	—	8·0	2·0
—	16	45·5	—	—
—	$16\frac{1}{4}$	—	3·0	2·25
—	$16\frac{5}{8}$	47·0	—	—
—	$16\frac{7}{8}$	—	1·33	1·83
—	$17\frac{1}{4}$	48·0	—	—
—	$18\frac{1}{4}$	—	0·75	2·1
—	$19\frac{1}{4}$	49·5	—	—
—	20	—	< 0·5	< 2·25
			> 0·33	> 1·5
—	$20\frac{3}{4}$	50·0	—	—
—	$25\frac{3}{4}$	—	0·25 (?)	^^^^^
—	$30\frac{3}{4}$	53·0	—	—
				a.m. 1·99
24·8	0 = $30\frac{3}{4}$	62·0 (?)	—	—
—	$31\frac{5}{16}$	—	8·0	1·5
—	$31\frac{1}{4}$	65·0	—	—

TABLE VIII.—*continued.*

P		τ		L		$\frac{\Delta L}{\Delta \tau}$		$\frac{\Delta L}{\Delta \tau} \times \tau$
—	..	$31\frac{7}{8}$	..	—	..	2·66	..	1·5
—	..	$31\frac{7}{8}$	..	66·0	..	—	..	—
—	..	$32\frac{7}{8}$	..	—	..	0·92	..	1·42
—	..	$33\frac{1}{8}$	..	67·5	..	—	..	—
—	..	$33\frac{1}{4}$	..	—	..	0·5	..	1·41
—	..	$34\frac{1}{4}$	..	68·0	..	—	..	—
—	..	$36\frac{1}{8}$	..	—	..	0·28	..	1·53
—	..	$38\frac{3}{8}$	..	69·25	..	—	..	—
—	..	$41\frac{1}{8}$	..	—	..	0·16	..	1·75
—	..	$44\frac{3}{4}$	..	70·25	..	—	..	—
							a.m.	**1·52**
44·8	..	0 = $45\frac{3}{4}$	..	90·0 (?)	..	—	..	2·62
—	..	$45\frac{7}{8}$	..	—	..	21·0	..	—
—	..	46	..	95·25	..	—	..	2·25
—	..	$46\frac{1}{4}$	..	—	..	6·0	..	—
—	..	$46\frac{1}{4}$	..	96·75	..	—	..	—
—	..	$46\frac{3}{4}$	..	—	..	3·0	..	2·25
—	..	$46\frac{7}{8}$	..	98·25	..	—	..	—
—	..	47	..	—	..	2·0	..	—
—	..	$47\frac{1}{4}$	..	99·25	..	—	..	2·5
—	..	$48\frac{1}{8}$	..	—	..	0·75	..	2·25
—	..	$50\frac{1}{4}$	..	101·5	..	—	..	—
—	..	$52\frac{1}{4}$	..	—	..	0·35	..	2·45
—	..	$55\frac{1}{4}$	..	103·25	..	—	..	—
—	..	$57\frac{3}{4}$	..	—	..	0·45 (?)	..	^^^^^
—	..	$60\frac{1}{4}$	..	105·5	..	—	..	—
							a.m.	**2 39**
74·8	..	0 = $60\frac{3}{4}$	..	127·5 (?)	..	—	..	—
—	..	$60\frac{7}{8}$	..	—	..	12·9	..	2·25
—	..	$60\frac{7}{8}$	..	132·0	..	—	..	—
—	..	61	..	—	..	3·5	..	2·63
—	..	$61\frac{1}{4}$	..	133·75	..	—	..	—
—	..	$61\frac{3}{4}$	..	—	..	2·0	..	3·0
—	..	$62\frac{1}{4}$	..	135·75	..	—	..	—
—	..	$63\frac{1}{4}$	..	—	..	0·92	..	3·0
—	..	$65\frac{1}{4}$	..	138·5	..	—	..	—
—	..	$67\frac{1}{4}$	..	—	..	0·5	..	3·75
—	..	$70\frac{1}{4}$	..	141·0	..	—	..	—
—	..	$72\frac{1}{4}$	..	—	..	0·35	..	4·4
—	..	$75\frac{1}{4}$	..	142·75	..	—	..	—
—	..	$80\frac{1}{4}$	..	—	..	0·275	..	5·5
—	..	$85\frac{1}{4}$	..	145·5	..	—	..	—
—	..	$90\frac{1}{4}$	..	—	..	0·225	..	6·75
—	..	$95\frac{1}{4}$	..	147·75	..	—	..	—
—	..	$100\frac{1}{4}$	..	—	..	0·175	..	7·0
—	..	$105\frac{1}{4}$	..	149·5	..	—	..	—
—	..	$110\frac{1}{4}$	..	—	..	0·175	..	8·75
—	..	$115\frac{1}{4}$	..	151·25	..	—	..	—
—	..	$120\frac{1}{4}$	..	—	..	0·15	..	9·0
—	..	$125\frac{1}{4}$	..	152·75	..	—	..	—
—	..	$130\frac{1}{4}$	..	—	..	0·025 (?)	..	—
—	..	$135\frac{1}{4}$	..	153·0	..	—	..	(?)
—	..	$137\frac{1}{4}$	..	153·0	..	—	..	—
44·8	..	0 = $137\frac{1}{4}$	..	135·5 (?)	..	—	..	—
—	..	$137\frac{1}{4}$	..	—	..	6·0	..	0·75
—	..	$137\frac{1}{4}$	..	134·0	..	—	..	—
—	..	138	..	—	..	2·0	..	0·75
—	..	$138\frac{1}{4}$	..	133·5	..	—	..	—
—	..	$138\frac{1}{16}$	..	—	..	0·46	..	0·75
—	..	$139\frac{1}{4}$	..	132·75	..	—	..	—
—	..	$141\frac{1}{2}$	..	—	..	$\begin{cases} <0·21 \\ >0·14 \end{cases}$	..	$\begin{cases} <0·86 \\ >0·57 \end{cases}$

TABLE VIII.—*continued.*

P		τ		L		$\frac{\Delta L}{\Delta \tau}$		$\frac{\Delta L}{\Delta \tau} \times \tau$
—	..	143½	..	132·0	..	—	..	—
—	..	147½	..	—	..	0·083	..	0·83
—	..	152¼	..	131·5	..	—	..	—
							a.m.	0·75
24·8	..	0=153	..	110·5 (?)	..	—	..	—
—	..	153½	..	—	..	8·0	..	1·0
—	..	153¾	..	108·5	..	—	..	—
—	..	153⅞	..	—	..	2·0	..	1·0
—	..	153⅞	..	107·5	..	—	..	—
—	..	154½	..	—	..	0·67	..	1·0
—	..	155½	..	106·5	..	—	..	—
—	..	156½	..	—	..	0·33	..	1·25
—	..	158½	..	105·5	..	—	..	—
—	..	160½	..	—	..	0·125	..	0·88
—	..	162½	..	105·0	..	—	..	—
—	..	164½	..	—	..	0·1	..	1·2
—	..	167½	..	104·5	..	—	..	—
							a.m.	1·05
14·8	..	0=168½	..	89·0 (?)	..	—	..	—
—	..	168¾	..	—	..	1·0	..	1·25
—	..	168⅞	..	86·5	..	—	..	—
—	..	168⅞	..	—	..	2·5	..	1·25
—	..	168⅞	..	85·25	..	—	..	—
—	..	169⅛	..	—	..	0·86	..	1·0
—	..	169½	..	84·5	..	—	..	—
—	..	170½	..	—	..	0·5	..	1·2
—	..	171¼	..	83·75	..	—	..	—
—	..	173¼	..	—	..	0·25	..	1·25
—	..	175¼	..	82·75	..	—	..	—
—	..	177¼	..	—	..	0·125	..	1·13
—	..	179¼	..	82·25	..	—	..	—
—	..	181	..	—	..	<0·16	..	<1·8
						>0·08	..	>0·91
—	..	182¾	..	82·0	..	—	..	—
							a.m.	1·22
7·8	..	0=183¼	..	68·25 (?)	..	—	..	—
—	..	183½	..	—	..	11·0	..	1·75
—	..	183⅝	..	64·75	..	—	..	—
—	..	183⅞	..	—	..	3·5	..	1·75
—	..	184	..	63·0	..	—	..	—
—	..	184½	..	61·5	..	1·2	..	1·65
—	..	185¼	..	61·5	..	—	..	—
—	..	187¼	..	—	..	0·4	..	1·8
—	..	190½	..	59·5	..	—	..	—
—	..	193½	..	—	..	0·82 (?)	..	(?)
—	..	197¼	..	57·25	..	—	..	—
							a.m.	1·74
4·8	..	0=198¼	..	53·0	..	—	..	—
—	..	198½	..	—	..	16·0	..	2·0
—	..	198½	..	49·0	..	—	..	—
—	..	198½	..	—	..	4·0	..	2·0
—	..	198⅞	..	47·0	..	—	..	—
—	..	199$\frac{1}{16}$	..	—	..	1·55	..	2·01
—	..	200	..	45·25	..	—	..	—
—	..	201½	..	—	..	0·58	..	1·97
—	..	203 × 60	..	43·5	..	—	..	—
							a.m.	2·00

TABLE IX.—*Experiment* 7 (January 5, 1905).

P	Δ P	$\frac{\Delta L}{\Delta \tau} \times \tau$	$\frac{\Delta L}{\Delta \tau} \times \frac{\tau}{\Delta P}$
4·8	—	—	—
—	3·0	—	—
7·8	—	1·56	0·52
—	7·0	—	—
14·8	—	1·99	0·28
—	10·0	—	—
24·8	—	1·52	0·15
—	20·0	—	—
44·8	—	2·39	0·12
—	30·0	—	—
74·8	—	2·44	0·081
—	30·0	—	—
44·8	—	0·75	0·025
—	20·0	—	—
24·8	—	1·05	0·052
—	10·0	—	—
14·8	—	1·22	0·12
—	7·0	—	—
7·8	—	1·74	0·25
—	3·0	—	—
4·8	—	2·00	0·67

TABLE X.—*Experiment* 8 (November 30, 1904).

P	τ	L.	$\frac{\Delta L}{\Delta \tau}$	$\frac{\Delta L}{\Delta \tau} \times \tau$
3·65	0·0 × 60	0·0	—	—
13·65	0·0 + ε	38·5 (?)	—	—
—	$\frac{1}{2}$	—	32·0	4·0
—	$\frac{3}{4}$	46·5	—	—
—	1	—	10·0	3·8
—	$\frac{3}{4}$	49·0	—	—
—	$\frac{1}{2}$	—	5·0	3·8
—	1	51·5	—	—
—	$1\frac{1}{2}$	—	3·0	4·5
—	2	54·5	—	—
—	$2\frac{1}{2}$	—	1·5	3·8
—	3	56·0	—	—
—	4	—	1·0	4·0
—	5	58·0	—	—
—	6	—	0·75	4·5
—	7	59·5	—	—
—	$8\frac{1}{2}$	—	0·5	4·25
—	10	61·0	—	—
—	11	—	1·4 (?)	^^^^^
—	12	64·0	—	—
—	$13\frac{1}{2}$	—	0·33	4·5
—	15	65·0	—	—
—	$17\frac{1}{4}$	—	0·22	4·0
—	$19\frac{1}{2}$	66·0	—	—
	^^^^^			a.m. 4·12
13·65	0 = $20\frac{1}{2}$	63·5	—	—
—	$21\frac{1}{4}$	—	1·33	1·0
—	22	65·5	—	—
—	23	—	0·5	1·25
—	24	66·5	—	—
—	27	—	0·17	1·1
—	30	67·5	—	—
—	$34\frac{3}{4}$	—	0·1	1·4
—	$39\frac{1}{2}$	68·5	—	—
				a.m. 1

TABLE X.—*continued.*

P		τ		L		$\frac{\Delta L}{\Delta \tau}$		$\frac{\Delta L}{\Delta \tau} \times \tau$
		^^^^^						
13·65	..	$0 = 40\frac{1}{4}$	..	64·5	..	—	..	—
—	..	$40\frac{3}{4}$	..	—	..	6·0	..	0·75
—	..	$40\frac{3}{4}$	..	66·0	..	—	..	—
—	..	$40\frac{3}{4}$	..	—	..	1·5	..	0·75
—	..	41	..	66·75	..	—	..	—
—	..	42	..	—	..	0·37	..	0·66
—	..	43	..	67·5	..	—	..	—
—	..	48	..	—	..	0·1	..	0·72
—	..	53	..	68·5	..	—	..	—
—	..	$57\frac{1}{4}$	..	—	..	$\left\{ \begin{array}{l} <0·06 \\ >0·03 \end{array} \right.$	..	$\begin{array}{l} <1·0 \\ >0·5 \end{array}$
—	..	$61\frac{1}{2}$	..	69·0	..	—	..	—
—	..	64	..	—	..	—	..	—
—	..	$65\frac{1}{2}$	..	70·0	..	0·2 (?)	..	^^^^^
							a.m.	**0·75**
		^^^^^						
13·65	..	$0 = 67\frac{1}{2}$	..	71·5	..	—	..	—
—	..	$68\frac{3}{4}$	..	—	..	0·52	..	0·6
—	..	70	..	72·5	..	—	..	—
—	..	75	..	—	..	0·1	..	0·7
—	..	80	..	73·5	..	—	..	—
—	..	85	..	—	..	$\left\{ \begin{array}{l} <0·05 \\ >0·025 \end{array} \right.$	..	$\begin{array}{l} <0·85 \\ >0·43 \end{array}$
—	..	90	..	74·0	..	—	..	—
							a.m.	**0·65**
		^^^^^						
13·65	..	$0 = 91\frac{3}{4}$	..	73·0	..	—	..	—
—	..	$91\frac{3}{4}$	..	—	..	4·0	..	0·5
—	..	92	..	74·0	..	—	..	—
—	..	$92\frac{1}{2}$	..	—	..	0·5	..	0·4
—	..	93	..	74·5	..	—	..	—
—	..	103	..	—	..	—	..	—
—	..	$112\frac{1}{2}$	..	75·5	..	$\left\{ \begin{array}{l} <0·05 \\ >0·04 \end{array} \right.$	..	$\begin{array}{l} <0·55 \\ >0·41 \end{array}$
							a.m.	**0·47**
3·65	..	$0 = 112\frac{1}{2} + \epsilon$	..	51·0 (?)	..	—	..	—
—	..	$112\frac{3}{4}$	..	—	..	26·0	..	3·25
—	..	$112\frac{3}{4}$	..	44·0	..	—	..	—
—	..	$112\frac{3}{4}$	..	—	..	8·0	..	3·0
—	..	113	..	42·0	..	—	..	—
—	..	$113\frac{1}{2}$	..	—	..	2·5	..	2·5
—	..	114	..	39·5	..	—	..	—
—	..	$114\frac{1}{2}$	..	—	..	1·5	..	3·0
—	..	115	..	38·0	..	—	..	—
—	..	$115\frac{1}{2}$	..	—	..	1·0	..	3·0
—	..	116	..	37·0	..	—	..	—
—	..	$118\frac{1}{2}$	..	—	..	0·5	..	3·0
—	..	121	..	34·5	..	—	..	—
—	..	$123\frac{1}{2}$	..	—	..	0·3	..	3·3
—	..	126	..	33·0	..	—	..	—
—	..	132	..	—	..	0·17	..	3·3
—	..	138	..	31·0	..	—	..	—
—	..	143	..	—	..	0·1	..	3·0
—	..	148	..	30·0	..	—	..	—
—	..	153	..	—	..	—	..	—
—	..	158	..	29·0	..	$\left\{ \begin{array}{l} >0·075 \\ <0·1 \end{array} \right.$	..	$\begin{array}{l} <4·0 \\ >3·0 \end{array}$
							a.m.	**3·12**

TABLE XI.—*Experiment* 9 (December 2, 1904).

P		τ		L		$\frac{\Delta L}{\Delta \tau}$		$\frac{\Delta L}{\Delta \tau} \times \tau$
3·65	..	0·0 × 60	..	0·0	..	—	..	—
33·65	..	0·0 + ε	..	95·0 (?)	..	—	..	
—	..	$\frac{1}{16}$	..	—	..	128·0	..	4·0
—	..	$\frac{1}{8}$	..	103·5	..	—	..	—
—	..	$\frac{3}{16}$	..	—	..	48·0	..	4·5
—	..	$\frac{1}{4}$	..	106·5	..	—	..	—
—	..	$\frac{5}{16}$	..	—	..	24·0	..	4·5
—	..	$\frac{3}{8}$	..	109·5	..	—	..	—
—	..	$\frac{7}{16}$	..	111·0	..	8)		
—	..	$\frac{1}{2}$	..	112·0	..	— 8·0	..	4·0
—	..	$\frac{9}{16}$	..	113·0	..	8)		
—	..	$\frac{5}{8}$	..	113·5	..	4)		
—	..	$\frac{11}{16}$	..	114·5	..	— 4·0	..	4·0
—	..	1	..	115·5	..	4)		
—	..	$1\frac{1}{8}$	..	116·5	..	2)		
—	..	$1\frac{1}{4}$	..	117·5	..	— 2·0	..	4·0
—	..	$1\frac{3}{8}$	..	118·5	..	2)		
—	..	$1\frac{1}{2}$	..	119·5	..	—	..	—
—	..	$1\frac{5}{8}$	..	—	..	1·0	..	4·0
—	..	$1\frac{3}{4}$	..	120·5	..	—	..	—
—	..	$1\frac{7}{8}$	..	—	..	0·75	..	4·8
—	..	2	..	123·5	..	—	..	—
—	..	$2\frac{1}{4}$	..	—	..	0·4	..	4·4
—	..	$2\frac{1}{2}$	..	125·5	..	—	..	—
—	..	$2\frac{3}{4}$	..	—	..	0·5 (?)	..	—
—	..	$3\frac{1}{4}$	..	127·0	..	—	..	—
—	..	$3\frac{1}{2}$	..	—	..	0·25	..	4·6
—	..	$3\frac{3}{4}$	..	128·0	..	—	..	—
—	..	$3\frac{7}{8}$	..	128·0	..	—	..	—
		^^^^^					a.m.	4·28
33·65	..	0 = $2\frac{1}{4}$	..	127·0	..	—	..	—
—	..	$2\frac{3}{8}$	..	—	..	3·3	..	1·25
—	..	$2\frac{1}{2}$	..	129·5	..	—	..	—
—	..	$2\frac{3}{4}$	..	—	..	1·0	..	1·25
—	..	3	..	130·5	..	—	..	—
—	..	$3\frac{1}{4}$	..	—	..	0·5	..	1·37
—	..	$3\frac{1}{2}$	..	131·5	..	—	..	—
—	..	$3\frac{3}{4}$	..	—	..	0·2	..	1·25
—	..	4	..	132·5	..	—	..	—
—	..	$4\frac{1}{4}$	..	—	..	0·03	..	1·26
—	..	$4\frac{1}{2}$	..	133·5	..	—	..	—
		^^^^^					a.m.	1·28
33·65	..	0 = $4\frac{1}{2}$	..	132·0	..	—	..	—
—	..	$4\frac{3}{4}$	..	—	..	0·2	..	1·0
—	..	5	..	134·0	..	—	..	—
—	..	$5\frac{1}{4}$	..	—	..	0·3	..	1·14
—	..	$5\frac{1}{2}$	..	135·0	..	—	..	—
—	..	$5\frac{3}{4}$	..	—	..	—	..	—
—	..	$5\frac{8}{4}$	..	136·0	..	0·09	..	1·0
—	..	$6\frac{1}{4}$	..	136·0	..	—	..	—
							a.m.	1·04

TABLE XI.—*continued.*

P		τ		L		$\frac{\Delta L}{\Delta \tau}$		$\frac{\Delta L}{\Delta \tau} \times \tau$
		^^^^^						
33·65	..	$0 = 61\frac{3}{4}$	..	134·5	..	—	..	—
	..	$62\frac{1}{2}$	..	—	..	1·0	..	0·88
—	..	$63\frac{1}{2}$	..	136·0	..	—	..	—
—	..	$65\frac{1}{2}$	..	—	..	0·25	..	0·94
—	..	$67\frac{1}{2}$	..	137·0	..	—	..	—
—	..	$74\frac{1}{2}$	..	—	..	0·7	..	0·80
	..	$81\frac{1}{2}$	..	138·0	..	—	..	—
—	..	$81\frac{3}{4}$	..	108·0	..	—	..	—
		^^^^^					a.m.	0·90
33·65	..	$0 = 83$	..	137·5	..	—	..	—
—	..	85	..	—	..	0·25	..	0·5
—	..	87	..	138·5	..	—	..	—
—	..	91	..	—	..	0·06	..	0·5
—	..	95	..	139·0	..	—	..	—
—	..	102	..	139·0	..	—	..	—
		^^^^^					a.m.	0·5
33·65	..	$0 = 103$	..	138·25	..	—	..	—
—	..	104	..	—	..	0·75	..	0·37
—	..	105	..	139·0	..	—	..	—
—	..	111	..	—	..	0·12	..	0·33
—	..	117	..	139·5	..	—	..	—
—	..	$122\frac{1}{2}$	..	139·5	..	—	..	—
							a.m.	0·35
3·65	..	$0 = 122\frac{7}{10}$	..	84·5 (C)	..	—	..	—
—	..	123	..	76·0	..	—	..	—
—	..	$123\frac{1}{2}$	..	—	..	12·0	..	4·2
—	..	$123\frac{1}{2}$	..	70·0	..	—	..	—
—	..	124	..	—	..	4·0	..	4·4
—	..	$124\frac{1}{2}$	..	66·0	..	—	..	—
—	..	125	..	—	..	2·0	..	4·2
—	..	$125\frac{1}{2}$	..	64·0	..	—	..	—
—	..	$126\frac{1}{2}$	..	—	..	1·25	..	4·5
—	..	$127\frac{1}{2}$	..	61·5	..	—	..	—
—	..	130	..	—	..	0·6	..	4·3
—	..	$132\frac{1}{2}$	..	58·5	..	—	..	—
—	..	$137\frac{1}{2}$	..	—	..	0·3	..	4·35
—	..	$142\frac{1}{2}$	..	55·5	..	—	..	—
—	..	$152\frac{1}{2}$	..	—	..	0·15	..	4·5
—	..	$162\frac{1}{2}$	..	52·5	..	—	..	—
—	..	170	..	—	..	<0·1	..	<4·7
—	..	$177\frac{1}{2}$	..	51·0	..	>0·08	..	>3·9
							a.m.	4·34

TABLE XII.—*Experiment* 10 (December 5, 1904).

P		τ		L		$\frac{\Delta L}{\Delta \tau}$		$\left[\frac{\Delta L}{\Delta \tau}\right]$ Extrapolation		$\frac{\Delta L}{\Delta \tau} \times \tau$
3·1	..	0·0 + 60	..	0·0	..	—	..	—	..	—
23·1	..	0·0 + ε	..	75·0	..	—	..	—	..	—
—	..	$\frac{1}{8}$	..	—	..	28·0	..	—	..	3·5
—	..	$\frac{1}{4}$	..	82·0	..	—	..	—	..	—
—	..	$\frac{3}{8}$	..	—	..	8·0	..	—	..	3·0
—	..	$\frac{1}{2}$	..	81·0	..	—	..	—	..	—
—	..	$\frac{3}{4}$	..	—	..	4·0	..	—	..	3·0
—	..	1	..	86·0	..	—	..	—	..	—
—	..	$1\frac{1}{2}$	..	—	..	2·5	..	—	..	3·75
—	..	2	..	88·5	..	—	..	—	..	—
—	..	$2\frac{1}{2}$	..	—	..	1·5	..	—	..	3·75
—	..	3	..	90·0	..	—	..	—	..	—
—	..	4	..	—	..	0·75	..	—	..	3·0
—	..	5	..	91·5	..	—	..	—	..	—
—	..	6	..	—	..	0·5	..	—	..	3·0
—	..	7	..	92·5	..	—	..	—	..	—
—	..	$8\frac{1}{2}$	..	—	..	0·35	..	—	..	3·0
—	..	$9\frac{7}{10}$	..	93·5						—
		^^^^^			..	2·3			a.m.	**3·25**
		11	..	96·0						
—	..	$13\frac{1}{2}$	..	—	..	0·2	..	0·21	..	3·25
—	..	16	..	97·0	..	—	..	—	..	—
—	..	18	..	—	..	0·25	..	0·18	..	3·25
—	..	20	..	98·0	..	—	..	—	..	—
—	..	$20\frac{1}{4}$	..	98·0						
		^^^^^			..	0·6	..	—	..	—
—	..	$21\frac{1}{2}$	..	99·0						
—	..	$23\frac{1}{4}$	..	—	..	0·11	..	0·14	..	3·25
—	..	26	..	99·5	..	—	..	—	..	—
—	..	$28\frac{1}{2}$	..	—	..	0·2	..	0·11	..	3·25
—	..	$31\frac{7}{10}$	..	100·5						
		^^^^^			..	0·36	..	—	..	—
—	..	$32\frac{1}{2}$	..	101·0						
—	..	37	..	—	..	0·0	..	0·09	..	3·25
—	..	$41\frac{1}{2}$	..	101·0						
		^^^^^			..	0·17	..	—	..	—
—	..	43	..	101·25						
—	..	47	..	—	..	0·0	..	0·07	..	3·25
—	..	$51\frac{1}{4}$	..	101·25						
		^^^^^			..	0·33	..	—	..	—
—	..	53	..	101·75						
—	..	$57\frac{1}{2}$	..	—	..	0·11	..	0·06	..	3·25
—	..	60	..	102·5	..	—	..	—	..	—

TABLE XIII.—*Experiment* 11 (December 29, 1904).

P	τ	L	$\frac{\Delta L}{\Delta \tau}$	$\frac{\Delta L}{\Delta \tau} \times \tau$
4·8	0·0 = 60	0·0	—	—
7·8	1	—	—	—
14·8	2	—	—	—
24·8	3	—	—	—
44·8	4	—	—	—
—	$5\frac{1}{2} - \epsilon$	105·0	—	—
74·8	$0 = 5\frac{1}{2}$	130·5 (?)	—	—
—	$5\frac{5}{10}$	—	22·5	2·25
—	6	135·0	—	—
—	$6\frac{1}{2}$	—	7·0	2·27
—	$6\frac{2}{4}$	136·75	—	—
—	$6\frac{3}{4}$	—	4·0	2·3
—	$6\frac{4}{4}$	137·75	—	—
—	$6\frac{5}{4}$	—	3·0	2·47
—	$6\frac{6}{4}$	138·5	—	—
—	$7\frac{1}{4}$	—	2·33	3·1
—	$7\frac{2}{4}$	140·25	—	—
—	$7\frac{3}{4}$	—	2·5	3·62
—	8	141·5	—	—
—	$8\frac{1}{2}$	—	—	—
—	9	142·75	$< 1·5$ $> 1·25$	$< 4·0$ $> 3·37$
—	10	—	0·87	3·67
—	11	144·5	—	—
—	12	—	0·62	3·87
—	13	145·75	—	—
—	14	—	0·5	4·1
—	15	146·75	—	—
—	17	—	0·44	4·8
—	19	148·5	—	—
—	21	—	0·37	5·62
—	23	150·0	—	—
—	25	—	0·31	5·94
—	27	151·25	—	—
—	31	—	0·25	6·25
—	35	153·25	—	—
—	40	—	0·20	6·8
—	45	155·25	—	—
—	50	—	0·15	6·6
—	55	156·75	—	—
—	60	—	0·12	6·77
—	65	158·0	—	—
—	65·5	158·0	—	—

TABLE XIV.—*Experiment* 12 (October 31, 1904).

τ	P	$\frac{\Delta P}{\Delta \tau}$	L	$\frac{\Delta L}{\Delta P}$	$\frac{\Delta L}{\Delta \tau}$
0·0 × 60	14·15	0·014	0·0		
1	15·02		—	1·08	0·0
			1·0		
2	15·90	0·015	2·75	—	0·0155
3	16·77	0·014	5·0	—	0·0230
4	17·63	0·014	7·5	—	0·0273
6	19·33	0·014	11·75	—	0·0212
8	20·96	0·013	15·5	—	0·0177
10	22·51	0·012	19·0	—	0·0162
15	26·09	0·011	26·75	—	0·0139
20	29·31	0·010	33·5	—	0·0118
25	32·20	0·009	39·5	—	0·0104
30	34·85	0·008	40·75	—	0·0087
35	37·25	0·007	49·75	—	0·0087
—	—		—	—	(?)
40	39·40	0·006	(?)	—	(?)
45	41·36	0·006	62·75	—	0·0054
50	43·23	0·006	66·25	—	0·0050
55	44·98	0·005	69·5	—	0·0046
60	46·61	0·005	72·5	—	0·0033
65	48·11	0·005	75·0	—	0·0030
70	49·47	0·004	77·25	—	0·0016
75	50·75	0·005	79·0	—	0·0013
80	52·10	0·007	80·75	1·06	0·0
126·5	72·00		—		
			101·75	—	—
	a.m.	0·0076			
126·5	72·00	0·034	101·75	0·24	0·0
127	70·96	0·035	101·5	0·24	0·0
128	68·87		101·0		

TABLE XV.—*Experiment* 13 (October 26, 1904).

τ	P	$\frac{\Delta P}{\Delta \tau}$	L	$\frac{\Delta L}{\Delta P}$	$\frac{\Delta L}{\Delta \tau}$
0·0 × 60	14·0		0·0	1·25	0·0
½	15·05	0·035			
			1·25		
1	16·1	0·035	2·75	—	0·004
1½	17·14	0·035	4·25	—	0·007
2	18·17	0·034	6·0	—	0·015
2½	19·19	0·034	8·0	—	0·024
3	20·21	0·034	11·0	—	0·058
				—	0·035
3½	21·22	0·034	13·25		
				—	0·029
4	22·22	0·033	15·50		
5	24·20	0·033	19·25	—	0·022
6	26·15	0·032	23·0	—	0·021
8	29·93	0·030	30·0	—	0·019
10	33·56	0·029	36·5	—	0·016
12	37·08	0·028	42·5	—	0·014
14	40·43	0·027	48·0	—	0·011
				—	0·009
16	43·69	0·027	53·25		
18	46·85	0·026	58·0	—	0·007
20	49·93	0·026	62·5	—	0·005
22	52·93	0·025	66·75	—	0·004
24	55·85	0·024	70·75	—	0·003
26	58·72	0·024	74·50	—	0·0013
		0·024		—	0·0012
28	61·55		78·25		
30	64·35	0·023	81·75	—	0·0005
		0·024		1·25	0·0
44½	85·32		108·0		
		a.m. 0·027			
44½	85·32	0·036	108·0		
46	82·68	0·038	107·75		
47	80·42	0·038	107·25	0·18	0·0
48	78·16	0·038	106·75		
49	75·90	0·038	106·25		
50	73·64	0·038	105·5		0·0057
52	69·12	0·038	104·25	—	0·0037
54	64·60	0·038	102·5	—	0·0078
56	60·09	0·037	100·75	—	0·0078
58	55·62	0·036	98·75	—	0·010
60	51·23	0·035	96·5	—	0·012
62	46·98	0·032	94·0	—	0·014
64	43·13	0·030	91·25	—	0·017
66	39·48	0·029	88·25	—	0·020
68	36·02	0·028	84·75	—	0·024
70	32·72	0·026	81·25	—	0·024
72	29·5	0·025	77·25	—	0·029
74	26·59	0·023	73·0	—	0·031
76	23·79	0·022	68·25	—	0·035
78	21·18	0·021	63·0	—	0·040
80	18·71	0·019	57·25	—	0·044
82	16·5	0·018	50·75	—	0·051
84	14·32	0·007	44·0	—	0·056
85	13·87	0·000	42·25	—	0·028
86	13·87		41·25	—	0·017
		a.m. 0·029			0·0063
88	13·87		40·25		
90	13·87	—	40·0	—	0·0042
95	13·87	—	39·0	—	0·0033
		—		—	0·0030
100	13·87		38·0		
105	13·87	—	37·25	—	0·0027
110	13·87	—	37·0	—	0·0008
120	13·87	—	37·0	—	0·000

TABLE XVI.—*Experiment* 14 (December 20, 1904). 1*st Tracing.*

τ	P	L	$\frac{\Delta}{\Delta P}$	$\frac{\Delta L}{\Delta \tau}$
0·0	10·75 + 0·0	0·0	0·44	0·0
0·14	0·42	0·25	—	2·3
0·28	0·85	0·75		3·0
0·43	1·29	1·25		4·0
0·57	1·73	2·0		4·0
0·71	2·16	2·75		5·8
0·85	2·59	3·75		7·6
1·0	3·04	5·0		8·8
1·14	3·47	6·5		11·8
1·28	3·89	8·25		14·8
1·42	4·32	10·5		13·4
1·71	5·20	14·75		11·6
2·0	6·08	18·5		10·9
2·28	6·93	22·0		10·5
2·56	7·78	25·25		9·8
2·71	8·24	27·0		9·25
3·66	11·13	37·0		8·5
4·61	14·02	46·0		7·6
5·56	16·91	54·5		6·6
6·51	19·80	62·0		5·8
7·46	22·69	68·75		5·0
8·41	25·57	74·75		4·25
9·36	28·47	80·0		3·7
11·26	34·24	89·5		3·45
13·16	40·02	98·5		3·01
15·06	45·80	106·75		2·67
16·96	51·57	111·25		2·27
18·86	57·35	121·0		1·88
20·76	63·13	127·0		1·5
22·66	68·91	132·25		1·08
23·61	71·80	134·5	0·42	0·85
24·08	73·23	135·5	—	—
29·78	10·75 + 73·23	139·75		

τ	P	L	$\frac{\Delta}{\Delta P}$	$\frac{\Delta L}{\Delta \tau} \times \tau$
0=31·35	10·75	38·25 (?)	—	—
31·82	—	—	—	1·91
32·3	—	34·5	—	—
33·1	—	—	—	1·84
34·2	—	32·5	—	—
36·1	—	—	—	1·95
38·0	—	31·0	—	—
40·35	—	—	—	1·92
42·7	—	30·0	—	—
45·1	—	—	—	2·14
47·5	—	29·25	—	—
49·87	—	—	—	1·95
52·25	—	28·75	—	—
54·62	—	—	—	2·11
57·0	—	28·25	—	—
59·37	—	—	—	1·83
61·75	—	28·0	—	—
600·0 by extrapolation	—	22·5	—	—
				a.m. 1·95

TABLE XVII.—*Experiment* 14 (December 20, 1904). *6th Tracing.*

τ	P	L	$\frac{\Delta L}{\Delta P}$	$\frac{\Delta L}{\Delta \tau}$
0·0	10·75 + 0·0	0·0	0·68	0·0
0·14	0·44	0·25	—	3·4
0·28	0·89	1·0	—	4·9
0·43	1·35	2·0	—	6·0
0·57	1·81	3·25	—	6·9
0·71	2·27	4·5	—	7·3
0·86	2·73	5 75	—	8·3
1·0	3·18	7·25	—	9·4
1·14	3·62	8·75	—	10·3
1·42	4·52	12·25	—	12·1
1·71	5·44	16·25	—	9·3
2·0	6·36	19·5	—	7·7
2·28	7·26	22·25	—	7·0
2·57	8·17	24·75	—	6·1
2·71	8·62	25·75	—	5·5
3·67	11·67	33·0	—	5·1
4·63	14·72	39·75	—	4·6
5·59	17·78	46·0	—	4·1
6·55	20·83	51·75	—	3·3
7·51	23·88	56·75	—	3·0
8·47	26·93	61·5	—	2·75
9·43	29·99	66·0	—	2·4
11·35	36·09	74·25	—	2·11
13·27	42·2	82·0	—	1·46
15·9	48·3	88·5	—	0·81
17·1	54 36	93·75	—	0·68
19·0	60·42	98·75	—	0·30
20·9	66·5	103·0	—	0·25
22·8	72·5	107·25		
24·7	78·58	111·25	0·61	—
25·17	80·04	112·25		
30·4	10·75 + 80·01	113·5	—	0·25

				$\frac{\Delta L}{\Delta \tau} \times \tau$
0 = 33·0	10·75	11·5 (?)	—	—
33 6	—	—	—	1·5
34·2	—	8·5	—	—
36·1	—	—	—	1·63
38·0	—	6·5	—	—
42·75	—	—	—	1·54
47·5	—	5·0	—	—
52·25	—	—	—	1·52
57·0	—	4·25	—	—
600·0 by extrapolation	—	0·0	—	—
			a.m.	1·56

TABLE XVIII.—*Experiment* 14 (December 20, 1904). 10*th Tracing.*

τ	P	L	$\frac{\Delta L}{\Delta P}$	$\frac{\Delta L}{\Delta \tau}$
0·0	10·75 + 0·0	0·0		
0·14	0·44	0·25	0·71	0·0
0·28	0·89	1·0	—	2·6
0·43	1·34	1·75	—	2·7
0·57	1·77	2·5	—	3·0
0·71	2·21	3·25	—	3·0
0·85	2·64	4·0	—	3·5
1·0	3·09	5·0	—	4·0
1·14	3·54	6·0	—	4·7
1·28	3·98	7·0	—	5·3
1·42	4·43	8·25	—	6·0
1·57	4·88	9·5	—	6·5
1·71	5·32	11·0	—	8·4
2·0	6·20	14·75	—	10·4
2·28	7·09	19·0	—	12·8
2·56	7·98	22·0	—	8·5
2·85	8·86	25·0	—	7·7
3·8	11·82	32·25	—	5·3
4·75	14·77	39·0	—	4·8
5·7	17·73	45·25	—	4·25
6·65	20·68	51·0	—	3·7
7·6	23·64	56·0	—	3·45
8·55	26·59	61·75	—	3·2
9·5	29·54	66·25	—	2·4
10·45	32·56	70·5	—	2·15
11·4	35·45	74·0	—	1·35
13·3	41·36	80·0	—	0·83
15·2	47·27	85·5	—	0·56
17·1	53·18	90·75	—	0·43
19·0	59·10	95·75	—	0·30
19·95	62·05	98·0		0·0
20·9	65·0	100·25		0·0
22·8	70·91	104·75	0·75	0·0
24·7	76·82	109·25		0·0
25·17	78·28	110·25		0·0
				$\frac{\Delta L}{\Delta \tau} \times \tau$
31·35	10·75 + 78·28	110·25	—	—
0 = 34·5	10·75	8·25	—	—
41·0	—	—	—	1·12
47·0	—	5·75	—	—
52·0	—	—	—	1·28
57·0	—	5·0	—	—
600·0 by extrapolation	—	1·25	—	—
				a.m. 1·20

TABLE XIX.—*Experiment* 14 (December 20, 1904).

		τ	$\frac{\Delta P}{\Delta \tau}$	$\frac{\Delta L}{\Delta P}$	$\frac{\Delta L}{\Delta \tau} \times \tau$
Exp.	1	24·08	3·04	0·42	1·95
	2	23·38	3·17	0·48	1·80
	3	25·51	3·17	0·51	1·70
	4	24·87	3·10	0·56	1·67
	6	25·17	3·18	0·61	1·56
	7	24·90	3·12	0·70	1·42
	9	25·36	3·11	0·75	1·12
	10	25·17	3·11	0·75	1·20
	11	26·22	3·06	0·77	1·13

TABLE XX.—*Experiment* 15 (December 15, 1904).

Exp.	1	59·85	1·17	0·32	1·89
	2	62·70	1·18	0·37	1·82
	5	63·65	1·17	0·53	1·57
	7	65·55	1·14	0·62	1·47
	9	63·65	1·20	0·64	1·30
	10	66·50	1·17	0·65	1·19

TABLE XXI.—*Experiment* 16 (December 14, 1904).

Exp.	1	97·85	0·65	0·34	1·88
	2	102·60	0·66	0·44	1·79
	3	104·50	0·66	0·51	1·66
	5	114·00	0·63	0·60	1·52
	6	114·95	0·64	0·64	1·41
	8	113·00	0·67	0·70	1·20

(*B*) *The Temperature Variable.*

TABLE XXII.—*Experiment* 1 (December 18, 1906).

τ	T	$\frac{\Delta T}{\Delta \tau}$	L	τ	T	$\frac{\Delta T}{\Delta \tau}$	L
0	12·0	—	0·00	64	32·0	0·33	− 0·20
2	12·0	—	0·00	68	33·3	0·35	− 0·40
4	12·0		+ 0·05	72	34·7	0·25	− 0·80
6	—		+ 0·05	74	35·2	0·35	− 1·10
8	—	0·50	+ 0·05	76	35·9	0·20	− 1·40
10	—		0·00	78	36·3	0·35	− 1·85
12	16·0		0·00	80	37·0	0·25	− 2·55
14	—	0·28	0·00	82	37·5	0·25	− 3·35
16	17·1		− 0·05	84	38·0	0·25	− 4·70
18	17·8	0·35	0·00	86	38·5	0·25	− 7·10
20	18·0	0·10	+ 0·05	88	39·0	0·25	− 10·35
22	—	0·15	+ 0·05	90	39·5	0·12	− 15·85
24	18·6		+ 0·05	92	—	—	− 22·65
28	19·4	0·20	+ 0·05	94	40·00	0·23	− 27·60
32	20·3	0·22	+ 0·05	96	—	—	− 32·30
36	21·3	0·25	+ 0·05	98	40·9	0·25	− 35·60
40	22·6	0·32	+ 0·05	100	—	—	− 38·10
44	24·0	0·35	+ 0·05	102	41·9	0·23	− 39·90
48	25·4	0·35	+ 0·05	106	42·8	0·25	− 42·10
52	27·0	0·40	0·00	110	43·8	0·23	− 43·10
56	28·8	0·45	− 0·05	114	44·7		− 43·60
60	30·4	0·40	− 0·10				

TABLE XXIII.—*Experiment* 2 (December 22, 1905).

τ	T	$\frac{\Delta T}{\Delta \tau}$	L	τ	T	$\frac{\Delta T}{\Delta \tau}$	L
0	—	—	0·00	46	33·0	—	− 0·20
2	12·0	—	+ 0·10	47	—	0·40	− 0·25
4	—	0·10	+ 0·15	48	33·8	—	− 0·30
6	12·4	0·30	+ 0·20	49	—	0·60	− 0·60
8	13·0	0·35	+ 0·25	50	35·0	0·50	− 0·70
10	13·7	0·20	+ 0·25	51	35·5	0·50	− 1·00
12	14·1	0·60	+ 0·25	52	36·0	1·10	− 1·40
14	15·3	0·55	+ 0·25	53	37·1	0·30	− 2·15
16	16·4	0·50	+ 0·25	54	37·4	0·30	− 2·95
18	17·4	0·50	+ 0·20	55	37·7	0·40	− 3·70
20	18·4	0·45	+ 0·20	56	38·1	0·60	− 5·00
22	19·3	0·50	+ 0·20	57	38·7	0·70	− 6·70
24	20·3	0·55	+ 0·15	58	39·4	0·50	− 9·75
26	21·4	0·60	+ 0·15	59	40·1	0·30	− 12·30
28	22·6	0·55	+ 0·20	60	40·4	0·20	− 14·70
30	23·7	0·55	+ 0·20	61	40·6	0·30	− 16·30
32	24·8	0·60	+ 0·20	62	40·9	0·40	− 19·20
34	26·0	0·45	+ 0·20	63	41·3	0·50	− 22·50
36	26·9	0·50	+ 0·20	64	41·8	1·00	− 27·20
38	27·9	1·00	+ 0·15	65	42·8	1·00	− 34·70
40	29·9	0·50	0·00	66	43·8	0·90	− 42·20
42	30·9	—	− 0·10	67	44·7	0·30	− 46·90
43	—	0·50	− 0·15	68	45·0	0·70	− 48·20
44	31·9	—	− 0·20	69	45·7	0·80	− 48·70
45	—	0·55		70	46·5		− 48·50

TABLE XXIV.—*Experiment* 3 (December 13, 1906).

τ	T	$\frac{\Delta T}{\Delta \tau}$	L	τ	T	$\frac{\Delta T}{\Delta \tau}$	L
0	10·8	0·05	0·00	57	—	0·45	+ 0·30
2	10·9	0·20	0·00	58	31·0	—	+ 0·25
4	11·3	0·35	0·00	59	—	—	+ 0·10
6	12·0	0·45	0·00	60	31·7	—	+ 0·10
8	12·9	0·15	+ 0·05	61	—	0·50	+ 0·05
10	13·2	0·35	+ 0·05	62	32·7	—	+ 0·05
12	13·9	0·35	+ 0·10	63	—	0·60	0·00
14	14·6	0·42	+ 0·15	64	34·9	—	− 0·10
16	—	—	+ 0·15	65	—	0·60	− 0·20
18	16·3	—	+ 0·15	66	36·1	—	− 0·65
20	—	0·55	+ 0·20	67	—	0·45	− 1·30
22	18·5	0·25	+ 0·25	68	37·0	—	− 2·20
24	19·0	0·45	+ 0·25	69	—	0·30	− 3·65
26	19·9	0·45	+ 0·30	70	37·6	—	− 4·70
28	20·8	0·30	+ 0·35	71	—	0·60	− 6·15
30	21·4	0·35	+ 0·35	72	38·8	—	7·65
32	22·1	0·45	+ 0·35	73	—	0·50	9·30
34	23·0	0·35	+ 0·35	74	39·8	—	11·15
36	23·7	0·35	+ 0·35	75	—	0·80	14·25
38	24·4	0·35	+ 0·35	76	41·4	—	17·65
40	25·1	0·40	+ 0·35	77	—	0·80	21·75
42	25·9	0·30	+ 0·35	78	43·0	—	27·40
44	26·5	0·40	+ 0·35	79	—	0·45	33·50
46	27·3	0·40	+ 0·35	80	43·9	—	39·85
48	28·1	0·35	+ 0·35	81	—	0·50	44·85
50	28·8	0·25	+ 0·35	82	44·9	—	48·75
52	29·3	0·15	+ 0·35	83	—	0·20	49·95
54	29·6	0·25	+ 0·35	84	45·3	0·30	50·45
56	30·1		+ 0·30	86	45·6		51·60

TABLE XXV.—*Experiment* 4 (December 13, 1906).

τ	T	$\frac{\Delta T}{\Delta \tau}$	L	τ	T	$\frac{\Delta T}{\Delta \tau}$	L
0	15·0		0·00	15	33·2	1·0	0·10
1	15·6	0·6	+ 0·05	16	34·5	1·3	0·25
2	17·0	0·4	+ 0·05	17	36·0	1·5	0·35
3	18·3	0·7	+ 0·10	18	37·2	1·2	2·50
4	19·3	1·0	+ 0·05	19	38·5	1·3	8·50
5	20·4	1·1	+ 0·10	20	40·0	1·5	16·75
6	22·3	1·9	+ 0·05	21	41·3	1·3	28·25
7	24·6	1·3	—	22	42·3	1·0	39·25
8	26·8	2·2	+ 0·05	23	43·0	0·7	46·75
9	28·5	1·7	0·00	24	43·6	0·6	51·25
10	30·0	1·5	− 0·05	25	44·3	0·7	52·65
11	—	0·5	− 0·05	26	45·0	0·7	52·80
12	31·0		− 0·05	27	45·7	0·7	52·65
13	31·6	0·4	− 0·05				
14	32·2	0·6	− 0·10				

TABLE XXVI.—*Experiment* 5 (December 24, 1906).

τ	T	$\frac{\Delta T}{\Delta \tau}$	L	τ	T	$\frac{\Delta T}{\Delta \tau}$	L
0	12·6		0·00	47	—	0·65	− 0·35
2	13·5	0·45	0·00	48	37·3	—	− 0·55
4	14·0	0·25	0·00	49	—	0·70	− 1·00
6	14·5	0·25	0·00	50	38·7	—	− 1·75
8	15·9	0·70	0·00	51	—	0·65	− 2·25
10	16·9	0·50	0.00	52	40·0	—	− 3·40
12	17·5	0·30	0·00	53	—	0·65	− 4·70
14	18·0	0·25	+ 0·05	54	41·3	—	− 6·30
16	18·5	0·25	+ 0·05	55	—	0·55	− 9·10
18	19·0	0·25	+ 0·05	56	42·4	—	− 13·10
20	20·0	0·50	+ 0·05	57	—	0·45	− 16·75
22	21·5	0·75	+ 0·05	58	43·3	—	− 20·50
24	23·0	0·75	+ 0·10	59	—	0·50	− 24·00
26	24·1	0·55	+ 0·10	60	44·3	—	− 28·00
28	25·8	0·85	+ 0·05	61	—	0·65	− 32·00
30	27·3	0·75	− 0·05	62	45·6	—	− 35·50
32	28·6	0·65	− 0·10	63	—	0·65	− 39·00
34	29·8	0·60	− 0·10	64	46·9	—	− 41·50
36	30·5	0·35	− 0·10	65	—	0·65	− 43·70
38	31·4	0·45	− 0·05	66	48·2	—	− 45·00
40	32·4	0·50	− 0·05	67	—	0·65	− 46·20
42	33·4	0·50	− 0·05	68	49·5	0·70	− 47·20
43	34·0	0·60	− 0·05	69	50·2	0·60	− 47·50
44	34·7	0·70	− 0·10	70	50·8		− 47·50
45	35·4	0·70	− 0·20				
46	36·0	0·60	− 0·30				

TABLE XXVII.—*Experiment* 6 (December 24, 1905).

τ	T	$\frac{\Delta T}{\Delta \tau}$	L	τ	T	$\frac{\Delta T}{\Delta \tau}$	L
0·0	15·9		0·00	16·0	39·6	2·0	5·25
2·0	21·2	2·6	+ 0·10	16·5	40·4	1·6	11·25
4·0	24·7	1·7	+ 0·10	17·0	41·6	2·4	19·75
6·0	26·3	0·8	+ 0·15	17·5	42·7	2·2	25·75
7·5	26·3	0·0	+ 0·20	18·0	43·8	2·2	31·75
8·5	26·3	0·0	+ 0·20	18·5	45·2	2·8	38·75
9·5	27·1	0·8	+ 0·25	19·0	46·0	1·6	42·65
10·5	28·1	1·0	+ 0·30	19·5	46·7	1·4	45·45
11·5	29·9	1·8	+ 0·30	20·0	47·3	1·2	46·75
12·5	32·3	2·4	+ 0·30	20·5	47·8	1·0	47·55
13·5	34·3	2·0	+ 0·30	21·5	48·3	0·5	47·65
14·5	36·2	1·9	+ 0·05	23·0	50·0	0·7	47·55
15·0	37·1	0·9	− 0·25				
15·5	38·6	3·0	− 1·75				

TABLE XXVIII.—*Experiment* 7 (December 1, 1906).

τ	T	$\frac{\Delta T}{\Delta \tau}$	L	τ	T	$\frac{\Delta T}{\Delta \tau}$	L
0	12·4		0·00	66	31·0	+ 1·10	− 1·70
5	12·4	+ 0·0	0·00	68	33·5	+ 1·25	− 2·50
10	12·4	+ 0·0	0·00	69	32·4	− 1·10	− 2·75
15	13·6	+ 0·24	0·00	70	29·5	− 2·90	− 2·70
17	14·4	+ 0·26	0·00	71	26·5	− 3·00	− 2·70
19	15·3	+ 0·45	0·00	72	25·0	− 1·50	− 2·65
20	15·8	+ 0·50	0·00	73	23·1	− 1·90	− 2·60
22	16·3	+ 0·25	0·00	74	21·7	− 1·40	− 2·50
24	17·0	+ 0·35	0·00	75	20·5	− 1·20	− 2·50
26	17·8	+ 0·40	0·00	76	21·8	+ 1·30	− 2·50
28	18·5	+ 0·35	0·00	77	24·5	+ 2·70	− 2·50
30	19·6	+ 0·55	0·00	78	27·0	+ 2·50	− 2·50
32	20·7	+ 0·55	0·00	79	29·0	+ 2·00	− 2·50
34	21·7	+ 0·50	0·00	80	30·5	+ 1·50	− 2·60
36	22·7	+ 0·50	0·00	81	31·5	+ 1·00	− 2·80
38	23·8	+ 0·55	− 0·05	82	32·5	+ 1·00	− 3·00
40	24·7	+ 0·45	− 0·10	83	33·2	+ 0·70	− 3·25
42	25·4	+ 0·35	− 0·15	84	34·0	+ 0·80	− 3·55
44	26·4	+ 0·50	− 0·30	85	34·4	+ 0·40	− 3·80
46	27·6	+ 0·60	− 0·35	86	33·6	− 0·80	− 4·00
48	29·2	+ 0·80	− 0·55	87	30·6	− 3·00	− 4·00
50	31·1	+ 0·85	− 0·95	88	27·4	− 2·20	− 4·00
52	32·5	+ 0·70	− 1·40	89	24·5	− 2·90	− 3·95
53	31·6	− 0·90	− 1·45	90	22·2	− 2·30	− 3·95
54	30·0	− 1·60	− 1·25	91	21·0	− 1·20	− 3·95
56	25·7	− 2·15	− 1·15	92	19·8	− 1·20	− 3·90
58	23·0	− 1·35	− 1·10	93	18·7	− 1·10	− 3·85
60	21·5	− 0·75	− 1·00	94	17·3	− 1·40	− 3·85
62	25·8	+ 2·25	− 1·05	95	16·1	− 1·20	− 3·80
64	28·8	+ 1·50	− 1·20	96	15·0	− 1·10	− 3·80

TABLE XXIX.—*Experiment* 8 (December 5, 1906).

τ	T	$\frac{\Delta T}{\Delta \tau}$	L	τ	T	$\frac{\Delta T}{\Delta \tau}$	L
0	13·0		+ 1·30	13	26·0		
1	19·2	+ 6·2	+ 1·30	14	29·5	+ 3·5	− 3·65
2	23·0	+ 3·8	+ 1·30	15	32·5	+ 3·0	− 4·00
3	26·3	+ 3·3	+ 1·30	16	35·0	+ 2·5	− 4·85
4	29·0	+ 2·7	+ 1·30	17	32·0	− 3·0	− 5·60
5	32·0	+ 3·0	+ 1·30	18	28·5	− 3·5	− 5·60
6	34·0	+ 2·0	+ 0·80	19	24·5	− 4·0	− 5·50
7	36·3	+ 2·3	− 0·70	20	28·0	+ 3·5	− 5·50
8	38·0	+ 1·7	− 2·95	21	32·0	+ 4·0	− 5·60
9	32·0	− 6·0	− 3·70	22	35·0	+ 3·0	− 6·20
10	28·0	− 4·0	− 3·70	23	38·5	+ 3·5	− 8·70
11	25·0	− 3·0	− 3·65	24	41·5	+ 3·0	−13·60
12	22·5	− 2·5	− 3·60				

TABLE XXX.—*Experiment* 9 (December 22, 1905).

τ	T	$\frac{\Delta T}{\Delta \tau}$	L	τ	T	$\frac{\Delta T}{\Delta \tau}$	L
0	24·4		+ 0·25	13·5	33·8	− 2·0	− 24·80
1	26·0	+ 1·6	+ 0·25	14	34·7	− 1·8	− 25·25
2	28·5	+ 2·5	+ 0·25	15	37·7	+ 3·0	− 26·40
3	31·4	+ 2·9	0·00	16	41·3	+ 3·6	− 32·25
4	34·8	+ 3·4	− 0·60	17	44·4	+ 3·1	− 43·80
4·25	35·4	+ 2·4	− 0·95	17·5	45·0	+ 1·2	− 46·00
4·5	36·3	+ 3·6	− 1·85	18	40·0	−10·0	− 49·80
4·75	37·1	+ 3·2	− 2·45	18·5	35·8	− 8·4	− 49·55
5	37·5	+ 1·6	− 3·00	19	34·9	− 1·8	− 49·50
6	33·2	− 4·3	− 2·40	19·5	36·5	+ 3·2	
7	31·4	− 1·8	− 2·30	20	37·5	+ 2·0	− 49·00
8	33·8	+ 2·4	− 2·30	21	40·2	+ 2·7	− 48·70
9	36·0	+ 2·2	− 2·30	21·5	42·0	+ 3·6	
10	37·9	+ 1·9	− 7·80	22	43·2	+ 2·4	− 48·50
11	39·5	+ 1·6	−20·30	23	45·9	+ 2·7	− 49·40
12	40·2	+ 0·7	−24·70	24	48·0	+ 2·1	− 50·20
12·5	38·0	− 2·2	−24·80	24·5	49·5	+ 3·0	− 51·00
13	34·8	− 6·4	−24·80	25	50·3	+ 1·6	− 51·00

[THE FOLLOWING TABLES SHOULD BE READ IN CONJUNCTION WITH CHAPTER II.]

TABLE XXXI.—*Experiment* 1 (September 15, 1899).

FIFTH EXTENSION CURVE.

τ	P	L	Tonus	$\left[C_2\left(a - \frac{\Delta L}{\Delta P}\right)\right]$	[Lgn. $C_1(P-\Delta)$]	Diff.
0·0 ..	0·0 + 1·86 ..	$0{\cdot}0 \times 10^{-3}$ ..	— ..	— ..	— ..	—
3·0 ..	2·53 ..	12·8 ..	$7{\cdot}8 \times 10^{-3}$ ..	− 0·850 ..	− 0·850 ..	0·000
6·2 ..	5·22 ..	30·8 ..	6·5 ..	− 0·708 ..	− 0·701 ..	+ 0·007
12·6 ..	10·61 ..	60·6 ..	5·2 ..	− 0·566 ..	− 0·562 ..	+ 0·004
25·4 ..	21·39 ..	102·1 ..	3·9 ..	− 0·425 ..	− 0·426 ..	− 0·001
33·5 ..	33·50 ..	121·3 ..	— ..	— ..	— ..	—

$\Delta = 0{\cdot}3$ $C_1 = 4{\cdot}93 \times 10^{-3}$ $C_2 = -109$.

TABLE XXXII.—*Experiment* 2 (December 6, 1899).

τ	P	L	Tonus	$\left[C_2\left(a - \frac{\Delta L}{\Delta P}\right)\right]$	[Lgn. $C_1(P-\Delta)$]	Diff.
0·0 ..	0·0 + 1·9 ..	$0{\cdot}0 \times 10^{-3}$ ..	— ..	— ..	— ..	—
3·0 ..	2·33 ..	13·1 ..	$8{\cdot}4 \times 10^{-3}$ ..	− 0·920 ..	− 0·928 ..	− 0·008
6·2 ..	4·81 ..	34·6 ..	7·0 ..	− 0·766 ..	− 0·755 ..	+ 0·011
12·6 ..	9·77 ..	67·9 ..	5·6 ..	− 0·613 ..	− 0·605 ..	+ 0·008
25·4 ..	19·69 ..	114·0 ..	4·2 ..	− 0·460 ..	− 0·466 ..	− 0·006
35·0 ..	27·14 ..	137·6 ..	— ..	— ..	— ..	—

$\Delta = 0{\cdot}7$ $C_1 = 4{\cdot}45 \times 10^{-3}$ $C_2 = -109{\cdot}5$

TABLE XXXIII.—*Experiment* 3 (November 11, 1899).

τ	P	L	Tonus	$\left[C_2\left(a - \frac{\Delta L}{\Delta P}\right)\right]$	[Lgn. $C_1(P-\Delta)$]	Diff.
0·0 ..	0·0 + 1·86 ..	$0{\cdot}0 \times 10^{-3}$ ..	— ..	— ..	— ..	—
3·0 ..	2·56 ..	17·0 ..	$7{\cdot}2 \times 10^{-3}$ ..	− 0·857 ..	− 0·857 ..	0·00
6·2 ..	5·28 ..	36·0 ..	6·0 ..	− 0·714 ..	− 0·708 ..	+ 0·006
12·6 ..	10·74 ..	64·9 ..	4·8 ..	− 0·571 ..	− 0·599 ..	− 0·028
25·4 ..	21·64 ..	104·0 ..	3·6 ..	− 0·428 ..	− 0·434 ..	− 0·006
30·9 ..	26·33 ..	116·0 ..	— ..	— ..	— ..	—

$\Delta = 0{\cdot}3$ $C_1 = 4{\cdot}69 \times 10^{-3}$ $C_2 = -119$

TABLE XXXIV.—*Experiment* 4 (November 10, 1899).

τ	P	L	Tonus	Plastic.
0·0	1·88 + 0·0	$0·00 \times 10^{-3}$	—	—
0·25	—	—	$0·92$ (?) $\times 10^{-3}$	—
0·5	0·29	0·27	—	—
0·75	—	—	3·67	—
1·0	0·58	1·33	—	—
1·25	—	—	—	$12·72 \times 10^{-3}$
1·5	—	—	4·59	—
1·75	—	—	—	12·94
2·0	1·16	3·99	—	—
2·5	—	—	8·25	13·09
3·0	1·74	7·98	—	—
4·6	—	—	—	11·90
6·2	3·60	20·22	9·00	—
9·4	—	—	—	10·68
12·6	7·31	43·62	7·20	—
19·0	—	—	—	8·53
25·4	14·74	75·55	5·40	—
33·4	—	—	—	7·07
41·4	24·01	103·74	3·60	—
42·4	24·57	105·20	—	—
	P = const.			
42·5	1·88 + 24·57	105·34	—	—
43·25	—	—	—	$0·77 \times 10^{-3}$
44·0	—	106·40	—	—
45·5	—	—	—	0·35
47·0	—	107·46	—	—
49·5	—	—	—	0·03
52·0	—	108·00	—	—
53·5	—	108·15	—	—
55·0	1·88 + 24·57	108·20	—	—
	Unloading of the muscle.			
55·0 + ϵ	1·88 + 0·0	13·78 (?)	—	—
56·5	—	12·77	—	—
57·0	—	—	—	$- 0·53 \times 10^{-3}$
57·5	—	12·24	—	—
58·5	—	11·97	—	—
59·0	—	—	—	− 0·27
59·5	—	11·70	—	—

TABLE XXXV.—*Experiment* 4 (November 10, 1899).

τ	$\left[C_2\left(a - \frac{\Delta L}{\Delta P}\right)\right]$	[Lgn. $C_1 (P - \Delta)$]	Diff.
3·0	− 0·756	− 0·760	− 0·004
6·2	− 0·605	− 0·597	+ 0·008
12·6	− 0·454	− 0·452	+ 0·002
25·4	− 0·302	− 0·313	− 0·011
41·4	− 0·227	− 0·220	+ 0·007

$\Delta = 0·4 \qquad C_1 = 13·2 \times 10^{-3} \qquad C_2 = - 84$

TABLE XXXVI.—*Experiment* 5 (December 2, 1899).

τ	P	L	Tonus	Plastic.	$\frac{dl}{d\tau} \times \tau$
0·0	3·90 + 0·0	$0·00 \times 10^{-3}$	—	—	
0·25	—	—	$1·52(?) \times 10^{-3}$	—	
0·5	0·53	0·81	—	—	
0·75	—	—	3·04	—	
1·0	1·06	2·42	—	—	
1·25	—	—	—	$13·06 \times 10^{-3}$	
1·5	—	—	4·06	—	
1·75	—	—	—	15·62	
2·0	2·12	6·71	—	—	
2·5	—	—	4·94	16·00	
3·0	3·18	11·95	6·00	—	
4·6	—	—	—	14·44	
6·2	6·57	28·49	5·00	—	
9·4	—	—	—	12·76	
12·6	13·36	56·98	4·00	—	
18·8	—	—	—	9·88	
25·0	26·40	91·91	3·50	—	
25·2	27·00	91·37	—	—	
	P = const.				
25·2	3·90 + 27·00	91·37	—	—	
25·35	—	—	—	1·79	
25·5	—	91·91	—	—	
26·0	—	—	—	1·07	
26·5	—	92·99	—	—	
27·5	—	93·26	—	—	
28·5	—	—	—	0·18	
29·5	—	93·52	—	—	
45·2	Section of the nervus tibialis.				
50·0	—	101·05	—	—	
54·0	—	—	—	0·27	$2·38 \times 10^{-3}$
58·0	—	103·20	—	—	
60·0	—	103·47	—	—	
62·5	—	—	—	0·15	2·60
65·0	3·90 + 27·00	104·27	—	—	
	Unloading of the muscle $\left[\text{Elast. } 3·09 \times 10^{-3}\right]$				
65·0 + ϵ	3·90 + 0·0	20·94	—	—	
65·5	—	—	—	− 0·54	
66·0	—	20·41	—	—	
66·75	—	—	—	− 0·18	
67·5	3·90 + 0·0	20·14	—	—	

TABLE XXXVII.—*Experiment* 6 (December 3, 1899).

τ	P	L	Tonus	Plastic.	$\frac{dl}{d\tau} \times \tau$
0·0	1·90 + 0·0	$0·0 \times 10^{-3}$	—	—	
0·375	—	—	$3·36 \times 10^{-3}$	—	
0·75	0·72	2·42	—	—	
0·875	—	—	4·30	$16·48 \times 10^{-3}$	
1	0·97	3·49	—	—	
1·25	—	—	5·60	15·88 (?)	
1·5	1·45	6·18	—	—	
1·75	—	—	6·72	16·90	
2·0	1·93	9·41	—	—	
2·25	—	—	7·13	17·36	
2·5	2·42	12·90	—	—	
2·75	—	—	6·72	15·36	
3·0	2·90	16·12	6·60	—	
4·6	—	—	—	14·51	
6·2	5·99	35·47	5·50	—	
9·4	—	—	—	12·60	
12·6	12·17	65·57	4·40	—	
19·0	—	—	—	9·74	
25·4	24·54	109·65	3·30	—	
26·27	—	—	—	7·90	
27·1	26·19	112·87	—	—	
	P = const.				
27·1	1·90 + 26·19	112·87	—	—	
27·5	—	113·14	—	—	
27·75	—	—	—	0·54	
28·0	—	113·41	—	—	
28·5	—	—	—	0·40	
29·0	—	113·81	—	—	
40·6	Section of the nervus tibialis.				
45·5	—	135·18			
46·0	—	—	—	1·07	$5·91 \times 10^{-3}$
46·5	—	136·26	—	—	
47·0	—	—	—	1·07	6·99
47·5	—	137·33	—	—	
48·0	—	—	—	0·89	6·70
48·5	—	138·22	—	—	
50·0	—	—	—	0·42	3·98
51·5	—	139·48	—	—	
53·75	—	—	—	0·30	4·01
56·0	—	141·10			
56·3	Contraction.				
61·5	—	142·17	—	—	
63·5	—	144·59	—	—	
67·0	—	144·86	—	—	
70·0	—	144·86	—	—	
77·3	1·90 + 26·19	144·98	—	—	
	Unloading of the muscle	[Elast. $3·57 \times 10^{-3}$]			
77·3 + ϵ	1·90 + 0·0	51·46	—	—	
79·0	—	44·48	—	—	
79·5	—	—	—	− 1·48	$-2·35 \times 10^{-3}$
80·0	—	43·00	—	—	
80·5	—	—	—	− 1·07	− 3·42
81·0	—	41·92	—	—	
81·5	—	—	—	− 0·66	− 2·77
82·0	—	41·26	—	—	
82·5	—	—	—	− 0·68	− 3·54
83·0	—	40·58	—	—	
83·5	—	—	—	− 0·54	− 3·35
84·0	—	40·04	—	—	
85·5	—	—	—	− 0·31	− 2·54
87·0	—	39·10	—	—	
98·5	—	—	—	− 0·17	− 3·57
110·0	—	35·21	—	—	

a.m. 3·09

TABLE XXXVIII.– *Experiment* 6.

FIRST EXTENSION CURVE OF THE ATONIC MUSCLE.

τ	P	L	Plastic.
132 = 0·0 ..	1·9 + 0·0 ..	0·00 × 10^{-3} ..	[Elast. 2·16 × 10^{-3}]
0·5 ..	0·5 ..	1·07 ..	
1·0 ..	1·0 ..	4·30 ..	—
1·25 ..	— ..	— ..	4·29 × 10^{-3}
1·5 ..	1·5 ..	7·52 ..	—
1·75 ..	— ..	— ..	4·83
2·0 ..	2·0 ..	11·02 ..	—
2·25 ..	— ..	— ..	5·91
2·5 ..	2·5 ..	15·05 ..	—
2·75 ..	— ..	— ..	3·76
3·0 ..	3·0 ..	18·01 ..	—
3·25 ..	— ..	— ..	3·22
3·5 ..	3·5 ..	20·69 ..	—
3·75 ..	— ..	— ..	3·22
4·0 ..	4·0 ..	23·38 ..	—
4·25 ..	— ..	— ..	3.22
4·5 ..	4·5 ..	26·07 ..	—
4·75 ..	— ..	— ..	2·68
5·0 ..	5·0 ..	28·49 ..	—
5·25 ..	— ..	— ..	2·48
5·5 ..	5·5 ..	30·91 ..	—
5·75 ..	— ..	— ..	2·14
6·0 ..	6·0 ..	33·06 ..	—
7·0 ..	— ..	— ..	2·14
8·0 ..	8·0 ..	41·66 ..	—
9·0 ..	— ..	— ..	2·14
10·0 ..	10·0 ..	50·26 ..	—
11·0 ..	— ..	— ..	1·74
12·0 ..	12·0 ..	58·05 ..	—
13·0 ..	— ..	— ..	1·33
14·0 ..	14·0 ..	65·04 ..	—
15·0 ..	— ..	— ..	1·07
16·0 ..	16·0 ..	71·49 ..	—
17·0 ..	— ..	— ..	0·80
18·0 ..	18·0 ..	77·40 ..	—
19·0 ..	— ..	— ..	0·20 (?)
20·0 ..	20·0 ..	82·77 ..	—
21·0 ..	— ..	— ..	0·72
22·0 ..	22·0 ..	87·88 ..	—
23·0 ..	— ..	— ..	0·26
24·0 ..	24·0 ..	92·72 ..	—
25·0 ..	— ..	— ..	0·13 (?)
26·0 ..	26·0 ..	97·29 ..	—
27·0 ..	— ..	— ..	0·13 (?)
28·0 ..	28·0 ..	101·86 ..	—
28·2 ..	28·2 ..	102·12 ..	—
	P = const.		
28·2 ..	1·9 + 28·2 ..	102·12 ..	—
30·1 ..	— ..	— ..	0·28
32·0 ..	1·9 + 28·2 ..	103·20 ..	—
	Unloading of the muscle.		
32·0 + ϵ ..	1·9 + 0·0 ..	32·25 (?) ..	—
34·0 ..	— ..	11·29 ..	—
35·5 ..	— ..	— ..	0·98
37·0 ..	— ..	8·33 ..	—
39·0 ..	— ..	— ..	0·47
41·0 ..	— ..	6·45 ..	—
43·0 ..	— ..	— ..	0·34
45·0 ..	— ..	5·11 ..	—
48·0 ..	— ..	— ..	0·18
51·0 ..	— ..	4·03 ..	—
56·0 ..	— ..	— ..	0·13
61·0 ..	— ..	2·69 ..	—
66·0 ..	— ..	— ..	0·08
71·0 ..	— ..	1·88 ..	—
77·5 ..	— ..	— ..	0·08
216 = 84·0 ..	1·9 + 0·0 ..	0·81 ..	—

$\frac{dl}{d\tau} \times \tau$
—
—
− 3·45 × 10^{-3}
—
− 3·29
—
− 3·70
—
− 2·88
—
− 3·22
—
− 2·75 } 3·29
—
− 3·82 }
—

a.m. 3·30

TABLE XXXVIII.—*continued.*

SECOND EXTENSION CURVE OF THE ATONIC MUSCLE.

τ	P	L	Plastic.
216 = 0·0 ..	1·9 + 0·0 ..	0·00 × 10^{-3} ..	[Elast. 2·09 × 10^{-3}]
0·5 ..	0·45 ..	0·94 ..	—
0·75 ..	— ..	— ..	3·74
1·0 ..	0·90 ..	3·76 ..	—
1·5 ..	— ..	— ..	4·02
2·0 ..	1·80 ..	9·67 ..	—
3·0 ..	— ..	— ..	3·75
4·0 ..	3·60 ..	20·96 ..	—
5·0 ..	— ..	— ..	3·22
6·0 ..	5·40 ..	31·17 ..	—
7·0 ..	— ..	— ..	2·95
8·0 ..	7·20 ..	40·85 ..	—
9·0 ..	— ..	— ..	2·41
10·0 ..	9·00 ..	49·45 ..	—
11·0 ..	— ..	— ..	1·87
12·0 ..	10·80 ..	56·97 ..	—
13·0 ..	— ..	— ..	1·60
14·0 ..	12·60 ..	63·96 ..	—
15·0 ..	— ..	— ..	1·33
16·0 ..	14·40 ..	70·41 ..	—
17·0 ..	— ..	— ..	1·07
18·0 ..	16·20 ..	76·32 ..	—
19·0 ..	— ..	— ..	0·93
20·0 ..	18·00 ..	81·97 ..	—
21·0 ..	— ..	— ..	0·66
22·0 ..	19·8 ..	87·07 ..	—
23·0 ..	— ..	— ..	0·38
24·0 ..	21·60 ..	91·64 ..	—
25·0 ..	— ..	— ..	0·41
26·0 ..	23·40 ..	96·21 ..	—
27·0 ..	— ..	— ..	0·26
28·0 ..	25·20 ..	100·51 ..	—
29·0 ..	— ..	— ..	0·26
30·0 ..	27·00 ..	104·81 ..	—
31·8 ..	28·62 ..	108·57 ..	—
	P = const.		
31·8 ..	1·9 + 28·62 ..	108·57 ..	—
33·5 ..	— ..	— ..	0·21
35·2 ..	1·9 + 28·62 ..	109·25 ..	—
	Unloading of the muscle.		
35·2 + ϵ ..	1·9 + 0·0 ..	36·14 (?) ..	—
37·0 ..	— ..	10·34 ..	—
38·0 ..	— ..	— ..	0·87
39·0 ..	— ..	8·60 ..	—
40·0 ..	— ..	— ..	0·31
41·0 ..	— ..	7·65 ..	—
42·0 ..	— ..	— ..	0·33
43·0 ..	— ..	6·99 ..	—
44·9 ..	— ..	— ..	0·27
45·0 ..	1·9 + 0·0 ..	6·45 ..	—

$\frac{dl}{d\tau} \times \tau$
—
—
$-2{\cdot}44 \times 10^{-3}$
—
$-1{\cdot}51$
—
$-2{\cdot}26$
—
$-2{\cdot}37$
—

a.m. 2·15

TABLE XXXIX.—*Experiment* 7 (December 1, 1899).

τ	P	L	Tonus	Plastic.	$\frac{dl}{d\tau} \times \tau$
0·0	3·95+0·0	$0·0 + 10^{-3}$	—	—	
0·25	—	—	$2·69 \times 10^{-3}$	—	
0·5	0·60	1·61	—	—	
0·75	—	—	4·93	$32·28 \times 10^{-3}$	
1·0	1·20	4·57	—	—	
1·5	—	—	7·17	32·00	
2·0	2·40	13·17	—	—	
2·25	—	—	7·94	—	
2·5	3·00	18·01	—	31·83	
2·75	—	—	8·96		
3·0	3·61	23·38	— 8·51		
3·25	—	—	8·06		
3·5	4·21	28·22	—	—	
5·2	6·25	43·27	—	—	
5·7	—	—	6·72		
6·2	7·45	51·33	— 6·49	—	
6·7	—	—	6·27	25·56	
7·2	8·65	58·86	—	—	
11·6	13·94	86·00	—	—	
12·1	—	—	4·48		
12·6	15·14	91·37	— 4·48	—	
13·1	—	—	4·48	21·18	
13·6	16·34	96·75	—	—	
20·0	24·04	125·24	—	—	
20·5	—	—	3·13		
21·0	25·24	129·00	— 3·02	—	
21·5	—	—	2·91	16·61	
22·0	26·44	132·49	—	—	
22·8	3·95 + 27·40	134·91	—	—	
	P = const.				
22·8	3·95 + 27·40	134·91	—	—	
23·9	—	—	—	0·98	
25·0	3·95 + 27·40	137·06	—	—	
	Unloading of the muscle.				
$25·0 + \epsilon$	3·95 + 0·0	64·23 (?)	—	—	
36·0	—	56·71	—	—	
42·0	—	56·71	—	0·00	
$46·0 - \epsilon$	—	56·71	—	—	
	Section of the nerve.				
46·0	—	−9·14	—	—	
46·125	—	—	—	10·75	$1·34 \times 10^{-3}$
46·25	—	−6·45	—	—	—
46·375	—	—	—	4·30	1·61
46·50	—	−5·37	—	—	—
46·75	—	—	—	2·70	2·02
47·0	—	−4·03	—	—	—
47·5	—	—	—	1·61	2·42
48·0	—	−2·42	—	—	—
49·0	—	—	—	0·54	1·61
50·0	—	−1·34	—	—	—
53·7	—	—	—	0·18	1·40
57·4	—	0·0	—	—	—

TABLE XL.—*Experiment* 7.

First Extension Curve of the Atonic Muscle.

T		P		L		Plastic.
57·4 = 0	..	3·95 + 0·00	..	0·00 × 10^{-3}	..	[Elast. 2·40 × 10^{-3}]
0·25	..	—	..	—	..	0·18
0·5	..	0·41	..	1·07	..	—
0·75	..	—	..	—	..	3·08
1·0	..	0·83	..	4·03	..	—
1·5	..	—	..	—	..	4·05
2·0	..	1·65	..	10·48	..	—
2·5	..	—	..	—	..	5·39
3·0	..	2·48	..	18·27	..	—
3·5	..	—	..	—	..	5·93
4·0	..	3·31	..	26·61	..	—
5·0	..	—	..	—	..	9·14
6·0	..	4·96	..	42·73	..	—
7·0	..	—	..	—	..	6·25
8·0	..	6·62	..	58·05	..	—
9·0	..	—	..	—	..	4·85
10·0	..	8·27	..	72·56	..	—
12·5	..	—	..	—	..	3·40
15·0	..	12·41	..	101·59	..	—
17·5	..	—	..	—	..	2·00
20·0	..	16·54	..	123·62	..	—
22·5	..	—	..	—	..	0·82
25·0	..	20·68	..	139·75	..	—
27·5	..	—	..	—	..	0·56
30·0	..	24·82	..	154·53	..	—
32·3	..	—	..	—	..	0·34
34·6	..	28·62	..	167·16	..	—
—	..	P = const.	..	—	..	—
34·6	..	3·95 + 28·62	..	167·16	..	—
35·8	..	—	..	—	..	0·45
37·0	..	3·95 + 28·62	..	168·24	..	—
				Unloading of the muscle		[Elast. 2·80 × 10^{-3}]
37·0 + ε		3·95 + 0·00	..	88·15	..	—

Second Extension Curve of the Atonic Muscle.

179·4 = 0	..	3·95 + 0·00	..	0·00 × 10^{-3}	..	[Elast. 2·40 × 10^{-3}]
0·25	..	—	..	—	..	0·00
0·5	..	0·67	..	1·34	..	—
0·75	..	—	..	—	..	0·40
1·0	..	1·33	..	2·69	..	—
1·5	..	—	..	—	..	0·54
2·0	..	2·67	..	5·91	..	—
2·5	..	—	..	—	..	1·63
3·0	..	4·00	..	10·21	..	—
3·5	..	—	..	—	..	4·05
4·0	..	5·33	..	16·93	..	—
5·0	..	—	..	—	..	4·17
6·0	..	8·00	..	30·64	..	—
7·0	..	—	..	—	..	3·78
8·0	..	10·66	..	43·54	..	—
9·0	..	—	..	—	..	2·16
10·0	..	13·33	..	53·21	..	—
12·5	..	—	..	—	..	1·84
15·0	..	20·00	..	75·79	..	—
17·5	..	—	..	—	..	0·98
20·0	..	26·66	..	94·06	..	—
22·1	..	—	..	—	..	0·65
24·2	..	32·26	..	108·04	..	—
		P = const.				
24·2	..	3·95 + 32·26	..	108·04	..	—
25·8	..	—	..	—	..	0·67
27·4	..	3·95 + 32·26	..	110·19	..	—
				Unloading of the muscle		[Elast. 2·43 × 10^{-3} (?)]
27·4 + ε		3·95 + 0·00	..	31·71 (?)	..	—

TABLE XLI.—*Experiment* 8 (December 4, 1900).

τ	P	L	Tonus	Plastic.
0·0	38 + 0·0	$0·0 \times 10^{-3}$	—	—
0·5	—	—	$0·45 \times 10^{-3}$	—
1·0	2·78	1·25	—	—
1·5	—	—	0·45	$4·3 \times 10^{-3}$
2·0	5·57	2·5	—	—
2·5	—	—	0·54	4·3
3·0	8·35	4·0	—	—
3·5	—	—	0·63	4·5
4·0	11·14	5·75	—	—
4·6	—	—	—	7·9
5·2	14·48	8·0	—	—
6·2	17·27	10·0	0·76	2·7 (?)
7·2	20·05	12·25	—	—
9·4	—	—	—	4·6
11·6	32·31	22·0	—	—
12·6	35·09	24·25	0·80	4·5
13·6	37·88	26·5	—	—
25·0	69·62	50·5	—	—
26·0	72·41	52·5	0·72	0·7 (?)
27·0	75·19	54·5	—	—
33·0	—	—	—	3·9
39·0	108·61	77·25	—	—
40·0	111·40	79·25	0·67	4·0
41·0	114·18	81·0	—	—
43·5	—	—	—	—
45·8	38+127·55	89·0	—	—
	P = const.			
45·8	38+127·55	89·0	—	—
48·0	—	—	—	0·38
50·4	38+127·55	90·75	—	—
			Unloading of the muscle	[Elast. $0·47 \times 10^{-3}$]
50·4 + ϵ	38+ 0·0	30·5	—	—

Elasticity of the muscle during the interval (0—1″) = $0·45 \times 10^{-3}$

Elasticity of the muscle at the unloading = $0·47 \times 10^{-3}$

τ	$C_2\left(a - \frac{\Delta L}{\Delta P}\right)$	Lgn. C_1 (P − Δ)	Diff.
12·6	− 1·347	− 1·351	− 0·004
26·0	− 1·212	− 1·213	− 0·001
40·0	− 1·128	− 1·131	− 0·003

Δ 0·6 $C_1 = 0·023 \times 10^{-5}$ $C_2 = -1684$

Length of the muscle, 11 cm.
Weight of the muscle, 37·5 grm.

TABLE XLII.—*Experiment* 8 (December 4, 1900).

τ	P	L	Tonus	Plastic.
0·0	38 + 0·0	$0·0 \times 10^{-3}$	—	—
0·5	—	—	$0·34 \times 10^{-3}$	—
1·0	2·94	1·0	—	—
1·5	—	—	0·43	$4·5 \times 10^{-3}$
2·0	5·88	2·25	—	—
2·5	—	—	0·51	4·4
3·0	8·82	3·75	—	—
3·5	—	—	0·68	4·9
4·0	11·76	5·75	—	—
4·6	—	—	—	5·0
5·2	15·28	8·5	—	—
6·2	18·22	10·75	0·81	6·6
7·2	21·16	13·25	—	—
11·6	34·09	23·25	—	—
12·6	37·03	25·5	0·72	4·6
13·6	39·97	27·5	—	—
19·5	—	—	—	4·4
25·0	73·47	51·75	—	—
26·0	76·41	53·75	0·64	2·4 (?)
27·0	79·35	55·5	—	—
33·0	—	—	—	4·1
39·0	114·62	77·0	—	—
40·0	117·56	78·75	0·60	4·4
41·0	120·50	80·5	—	—
45·5	—	—	0·60	—
49·9	38 + 146·61	95·75	—	—
	P = const.			
49·9	38 + 146·61	95·75	—	—
52·0	—	—	—	0·7
54·0	38 + 146·61	97·25	—	—
			Unloading of the muscle	[Elast. $0·39 \times 10^{-3}$]
54·0 + ϵ	38 + 0·0	39·5	—	—

Elasticity of the muscle during the interval $(0—1^{s}) = 0·34 \times 10^{-3}$

Elasticity of the muscle at the unloading $= 0·39 \times 10^{-3}$

τ	$C_2\left(a - \frac{\Delta L}{\Delta P}\right)$	Lgn. C_1 (P − Δ)	Diff.
6·2	− 1·411	− 1·400	+ 0·011
12·6	− 1·254	− 1·261	− 0·007
26·0	− 1·115	1·128	− 0·013
40·0	− 1·045	− 1·042	+ 0·003

$\Delta = 0·6$ $\quad C_1 = 0·034 \times 10^{-3}$ $\quad C_2 = -1742$

TABLE XLIII.—*Experiment* 9 (December 6, 1900).

τ	P	L	Tonus	Plastic.
0·0	38 + 0·0	$0·0 \times 10^{-3}$	—	—
0·5	—	—	$0·20 \times 10^{-3}$	—
1·0	2·51	0·5	—	—
1·5	—	—	0·40	$4·4 \times 10^{-3}$
2·0	5·02	1·5	—	—
2·5	—	—	0·50	4·5
3·0	7·53	2·75	—	—
3·5	—	—	0·50	4·3
4·0	10·04	4·0	—	—
4·6	—	—	—	4·5
5·2	13·06	5·75	—	—
6·2	15·57	7·25	0·60	9·4
7·2	18·08	8·75	—	—
11·6	29·13	16·25	—	—
12·6	31·64	18·0	0·70	4·3
13·6	34·15	19·75	—	—
25·0	62·78	38·75	—	—
26·0	65·29	40·25	0·60	3·8
27·0	67·80	41·75	—	—
53·0	133·08	77·0	—	—
54·0	135·49	78·25	0·50	3·3
55·0	138·11	79·5	—	—
57·9	38 + 145·40	83·0	—	—
	P = const.			
57·9	38 + 145·40	83·0	—	—
61·0	—	—	—	0·6
64·0	38 + 145·40	86·5	—	—
		Unloading of the muscle		[Elast. $0·26 \times 10^{-3}$]
64·0 + ϵ	38 + 0·0	48·0	—	—

Elasticity of the muscle during the interval (0—1″) = $0·20 \times 10^{-3}$

Elasticity of the muscle at the unloading = $0·26 \times 10^{-3}$

τ	$C_2\left(a - \frac{\Delta L}{\Delta P}\right)$	Lgn. $C_1 (P - \Delta)$	Diff.
12·6	− 0·968	− 0·968	0·0
26·0	− 0·830	− 0·829	+ 0·001
54·0	− 0·692	− 0·691	+ 0·001

$\Delta = 0·5$ $C_1 = 0·189 \times 10^{-3}$ $C_2 = -1383$

Length of the muscle, 10·5 cm.
Weight of the muscle, 34·2 grm.

TABLE XLIV.—*Experiment* 9 (December 6, 1900).

τ	P	L		Tonus	Plastic.
0·0	38 + 0·0	0·0 × 10^{-3}		—	—
0·5	—	—		0·26 × 10^{-3}	—
1·0	2·9	0·75		—	—
1·5	—	—		0·43	4·4 × 10^{-3}
2·0	5·8	2·0		—	—
2·5	—	—		0·52	4·5
3·0	8·7	3·5		—	—
3·5	—	—		0·52	4·4
4·0	11·6	5·0		—	—
4·6	—	—		—	4·4
5·2	15·08	7·0		—	—
6·2	17·98	9·0		0·69	4·7
7·2	20·88	11·0		—	—
9·5	—	—		—	4·4
11·6	33·63	19·25	0·69		
12·6	36·53	21·25	0·52	0·60	4·2
13·6	39·43	22·75			
25·0	72·49	41·5		—	—
26·0	75·39	43·0		0·52	3·7
27·0	78·29	44·5		—	—
33·0	—	—		—	3·5
39·0	113·08	61·75	0,52		
40·0	115·98	63·25	0·43	0·47	3·4
41·0	118·88	64·5			
45·0	—	—		0·42	—
48·7	38 + 141·21	74·0		—	—
P = const.					
48·7	38 + 141·21	—		—	—
52·0	—	—		—	0·5
54·8	38 + 141·21	—		—	—
				Unloading of the muscle	[Elast. 0·27 × 10^{-3}]
54·8 + ε	38 + 0·0	39·0		—	—

Elasticity of the muscle during the interval (0—1″) = 0·26 × 10^{-3}

Elasticity of the muscle at the unloading = 0·27 × 10^{-3}

τ	$C_2\left(a - \frac{\Delta L}{\Delta P}\right)$	Lgn. C_1 (P − Δ)	Diff.
6·2	− 1·126	− 1·122	+ 0·004
12·6	− 0·979	− 0·986	− 0·007
26·0	− 0·849	− 0·848	+ 0·001
40·0	− 0·767	− 0·766	+ 0·001

Δ = 0·5 C_1 = 0·148 × 10^{-3} C_2 = − 1632.

TABLE XLV.—*Experiment* 10 (April 6, 1902).

τ	P	L	Tonus
0·0	50 + 0	0·0 × 10^{-3}	—
1·25	89	0·0	0·05 × 10^{-3}
3·75	267	12·9	0·05
6·25	445	19·3	—
6·5	463	25·8	—
9·0	641	38·7	0·07
11·5	819	51·6	—
11·75	836	51·6	—
14·25	1014	64·4	0·09
16·75	1192	83·8	—
20·0	1424	109·6	—
22·5	1602	128·9	0·11
25·0	1779	148·2	—
32·5	2313	212·7	—
35·0	2491	232·0	0·13
37·5	2669	257·7	—
41·0	2918	290·0	—
43·5	3096	341·6	0·25
46·0	3274	380·0	—
52·0	3701	470·5	—
54·5	3879	502·7	0·18
57·0	4057	534·9	—
65·5	4662	625·2	—
68·0	4840	644·5	0·11
70·5	5018	663·8	—
82·5	5872	721·8	—
85·0	6050	728·3	0·04
87·5	50 + 6228	734·7	—
	P = const.		
97·5	50 + 6228	734·7	—

TABLE XLVI.—*Experiment* 11 (March 16, 1902).

τ	P	L	Tonus
0·0	50 + 0	0·0 × 10^{-3}	0·14 × 10^{-3}
1·25	92	12·9	
3·75	275	51·6	0·18
6·25	458	77·3	—
6·5	477	90·2	—
9·0	660	141·8	0·30
11·5	843	199·8	—
11·75	862	206·2	—
14·25	1045	270·7	0·39
16·75	1228	348·0	—
20·0	1467	425·4	—
22·5	1650	476·9	0·28
25·0	1833	528·5	—
25·5	1870	541·4	—
28·0	2053	580·1	0·21
30·5	2237	618·7	—
32·5	2383	644·5	—
35·0	2567	670·3	0·15
37·5	2750	696·1	—
41·0	3007	728·3	—
43·5	3190	741·2	0·07
46·0	3373	754·1	—
52·0	3813	773·4	—
54·5	3997	773·4	0·00
57·0	50 + 4180	773·4	—
	P = const.		
62·0	50 + 4180	773·4	—

TABLE XLVII.—*Experiment* 12 (April 6, 1902).

τ	P	L	Tonus
0·0	50 + 0	0·0 × 10^{-3}	—
1·25	70	8·5	0·16 × 10^{-3}
3·75	210	34·2	0·24
6·25	350	68·4	—
6·5	364	77·0	—
9·0	504	128·3	0·40
11·5	643	188·2	—
11·75	657	196·7	—
14·25	797	273·7	0·55
16·75	937	350·7	—
20·0	1119	470·4	—
22·5	1259	564·4	0·73
25·0	1399	675·7	—
32·5	1818	1017·8	—
35·0	1958	1128·9	0·73
37·5	2098	1223·0	—
41·0	2294	1334·2	—
43·5	2434	1419·8	0·61
46·0	2574	1505·3	—
52·0	2909	1684·9	—
54·5	3049	1753·3	0·49
57·0	3189	1821·7	—
65·5	3665	2027·0	—
68·0	3805	2086·9	0·37
70·5	3944	2129·6	—
82·5	4616	2249·4	0·18* (?)
85·0	50 + 4756	2275·0	
	P = const.		
90·0	50 + 4756	2275·0	—

* The movement of the foot is checked in the malleolar joint.

TABLE XLVIII.—*Experiment* 13 (March 9, 1902).

τ	P	L	Tonus
0·0	50 + 0	0·0 × 10^{-3}	—
1·25	51	25·7	0·51 × 10^{-3}
3·75	152	77·0	0·46
6·25	253	119·7	—
6·5	264	119·7	—
9·0	365	171·1	0·51
11·5	466	222·4	—
11·75	476	230·9	—
14·25	578	290·8	0·59
16·75	679	350·7	—
20·0	811	436·2	—
22·5	912	504·6	0·68
25·0	1013	573·0	—
32·5	1317	778·3	—
35·0	1419	855·3	0·76
37·5	1520	932·3	—
41·0	1662	1034·9	—
43·5	1763	1120·4	0·84
46·0	1865	1205·9	—
52·0	2108	1394·1	—
54·5	2209	1462·5	0·64
57·0	2311	1522·4	—
65·5	2655	1673·4	—
68·0	2757	1736·2	0·42
70·5	2858	1779·0	—
82·5	3344	1924·4	—
85·0	3446	1950·0	0·21
87·5	50 + 3547	1967·1	—
	P = const.		
90·0	50 + 3547	1967·1	—

TABLE XLIX.—*Experiment* 14 (November 29, 1899).

τ		P	L	Tonus	Plastic.
0·0		1·9 + 0·0	$0·00 \times 10^{-3}$	—	—
0·25		—	—	$2·28 \times 10^{-3}$	—
0·5		0·51	1·07	—	—
0·75		—	—	2·36	$20·60 \times 10^{-3}$
1·0		1·03	2·28	—	—
1·25		—	—	3·44	19·35
1·5		1·53	4·03	—	—
1·75		—	—	4·22	19·85
2·0		2·05	6·18	—	—
2·25		—	—	4·74	19·44
2·5		2·56	8·60	—	—
2·75		—	—	7·38	21·64
3·0		3·07	12·36	—	—
3·25		—	—	6·32	20·17
3·5		3·58	15·59	—	—
4·8		—	—	—	18·72
6·2		6.34	31·44	5·25	—
8·4					
10·0	Stimulation of pretibial muscles	10·30	52·94	—	—
10·5		—	—	5·22	22·71
11·0		11·25	58·32	—	—
11·5		—	—	5·22	16·32
12·0		12·28	63·69	—	—
12·4					
14·5		14·83	74·98	—	—
14·75		—	—	3·69	—
15·0		15·35	76·86	—	14·21
15·25		—	—	3·16	—
15·5		15·86	78·47	—	—
15·75		—	—	3·16	—
16·0		16·37	80·09	—	—
17·9					
18·0	Stimulation of pretibial muscles	18·40	85·73	—	—
18·5		—	—	4·17	14·20
19·0		19·43	90·03	—	—
19·5		—	—	3·65	13·53
20·0		20·46	93·79	—	—
21·4					
25·4		25·98	109·92	2·10	—
27·4		—	—	2·15	11·26
29·3		29·97	118·52	—	—
	P = const.				
29·3		1·9 + 29·97	118·52	—	—
29·6		—	—	—	0·38
30·0		—	118·79	—	—
31·0		—	—	—	0·27
32·0		—	119·33	—	—
33·0		1·9 + 29·97	119·60 (?)	—	—
	Unloading of the muscle.				
33·0 + ϵ		1·9 + 0·0	(?)	—	—

TABLE L.—*Experiment* 15 (November 26, 1899).

τ		P	L	Tonus	Plastic.
0·0		1·9 + 0·00	0·00 × 10^{-3}	—	—
0·5		0·54	1·61	3·45 × 10^{-3}	—
0·75		—	—	3·91	16·77 × 10^{-3}
1·0		1·09	3·76	—	—
1·25		—	—	5·48	—
1·5		1·63	6·72	—	17·93
1·75		—	—	7·33	—
2·0		2·18	10·75	—	—
2·25		—	—	6·97	17·61
2·5		2·72	11·51	—	—
2·75		—	—	6·59	17·21
3·0		3·27	18·14	—	—
3·3		—	—	—	—
3·5		3·81	23·11	—	—
3·75		—	—	6·97	16·80
4·0		4·35	26·87	—	—
4·25		—	—	7·47	17·31
4·5		4·90	30·91	—	—
4·75		—	—	7·97	16·45
5·0		5·44	34·67	—	—
5·5		—	—	6·90	16·29
6·0		6·53	42·19	—	—
6·5		—	—	5·92	14·96
7·0		7·62	48·64	—	—
7·5		—	—	6·65	15·55
8·0		8·71	55·90	—	—
8·25		—	—	5·97	15·63
8·5		9·25	59·12	—	—
8·6		—	—	—	—
11·6		12·63	77·13	—	—
12·1		—	—	4·68	12·68
12·6		13·72	82·24	—	—
13·1		—	—	4·68	12·57
13·6		14·81	87·34	—	—
22·0		23·95	120·94	—	—
22·5		—	—	3·70	10·68
23·0		25·04	124·97	—	—
23·5		—	—	3·70	10·62
24·0		26·13	129·00	—	—
24·4		—	—	3·70	—
24·8		27·0	132·22	—	—
	P = const.				
24·8		1·9 + 27·0	132·22	—	—
26·0		—	132·76	—	—
26·5		—	—	—	0·54
27·0		—	133·30	—	—
27·3		—	—	—	0·45
27·6		1·9 + 27·0	133·57	—	—
			Unloading of the muscle		[Elastic 3·58 × 10^{-3}]
27·6 + ε		1·9 + 0·00	36·82	—	—
28·5		—	10·75 (?)	—	—
29·0		—	—	—	− 2·15
29·5		—	8·60	—	—
30·5		—	—	—	− 1·07
31·5		—	6·45	—	—
32·0		—	—	—	− 0·44
32·5		1·9 + 0·00	5·91	—	—

Stimulation of pretibial muscle

TABLE LI.—*Experiment* 16 (October 25, 1909).

7		P		L		Tonus	
0·0	..	1·96 + 0·0	..	0·00 × 10^{-3}	..	—	
1·0	..	1·01	..	1·65	..	+ 2·86 × 10^{-3}	
2·0	..	2·02	..	5·77	..	—	
2·5	..	—	..	—	..	+ 5·71	
3·0	..	3·03	..	11·54	..	—	
3·5	..	—	..	—	..	+ 7·07	
4·0	..	4·04	..	18·68	..	—	
4·5	..	—	..	—	..	+ 7·07	
5·0	..	5·05	..	25·83	..	—	
5·4	..	—	..	—	..	+ 6·04	
5·5	..	5·55	..	28·85	..	—	
5·55	..	—	..	—	..	+10·00	
5·6	..	5·66	..	29·95	..	—	
5·65	..	—	..	—	..	+ 5·00	
5·7	..	5·76	..	30·50	..	—	
5·75	..	—	..	—	..	+ 5·50	
5·8	..	5·86	..	31·05	..	—	
5·85	..	—	..	—	..	+ 2·70	
5·9	..	5·96	..	31·32	..	—	
5·95	..	—	..	—	..	+ 0·60	
6·0	..	6·06	..	31·38	..	—	
6·05	..	—	..	—	..	− 3·30	
6·1	..	6·16	..	31·05	..	—	
6·15	..	—	..	—	..	− 8·30	
6·2	..	6·26	..	30·22	..	—	
6·25	..	—	..	—	..	0·00	
6·3	..	6·36	..	30·22	..	—	
6·35	..	—	..	—	..	+ 2·80	
6·4	..	6·46	..	30·58	..	—	
6·45	..	—	..	—	..	+ 5·50	
6·5	..	6·56	..	31·05	..	—	
6·55	..	—	..	—	..	+ 2·70	
6·6	..	6·67	..	31·32	..	—	
6·65	..	—	..	—	..	0·00	
6·7	..	6·77	..	31·32	..	—	
6·75	..	—	..	—	..	0·00	
6·8	..	6·87	..	31·32	..	—	
6·85	..	—	..	—	..	+ 2·80	
6·9	..	6·97	..	31·60	..	—	
6·95	..	—	..	—	..	+ 5·50	
7·0	..	7·07	..	32·15	..	—	
7·05	..	—	..	—	..	+ 8·20	
7·10	..	7·17	..	32·97	..	—	
7·15	..	—	..	—	..	+11·00	
7·2	..	7·27	..	34·07	..	—	
7·25	..	—	..	—	..	+13·70	
7·3	..	7·37	..	35·44	..	—	
7·35	..	—	..	—	..	+13·80	
7·4	..	7·47	..	36·82	..	—	
7·45	..	—	..	—	..	+10·00	
7·50	..	7·57	..	37·92	..	—	
7·55	..	—	..	—	..	+10·90	
7·60	..	7·68	..	39·01	..	—	
7·65	..	—	..	—	..	+11·00	
7·70	..	7·78	..	40·11	..	—	
7·75	..	—	..	—	..	+ 8·30	
7·80	..	7·88	..	40·94	..	—	
7·85	..	—	..	—	..	+ 5·50	
7·9	..	7·98	..	41·49	..	—	
7·95	..	—	..	—	..	+ 5·50	
8·0	..	8·08	..	42·04	..	—	
8·5	..	—	..	—	..	+ 5·20	
9·0	..	9·09	..	47·26	..	—	
9·5	..	—	..	—	..	+ 4·60	
10·0	..	10·10	..	51·93	..	—	
10·5	..	—	..	—	..	+ 3·80	
11·0	..	11·11	..	55·77	..	—	
11·5	..	—	..	—	..	+ 3·50	
12·0	..	12·12	..	59·35	..	—	
12·1	..	12·22	..	—	..	—	

Stimulation of pretibial muscles

[THESE TABLES SHOULD BE READ IN CONJUNCTION WITH CHAPTER III.]

THE ISOTONIC CONTRACTION.

TABLE LII.—*Experiment* 6.—TIGERSTEDT and WILLHARD.

No.	P	$C_1(P-\Delta)$	L. obs.	L. calc.	Const.
1	65	6·9	1·8	2·2	$\Delta = 62$ $C_1 = 2{\cdot}30$ $C_2 = 0{\cdot}90$
2	70	18·4	4·4	4·0	
3	75	29·9	5·4	5·2	
4	80	41·5	6·1	6·0	
5	85	53·0	6·6	6·45	
6	90	64·5	6·65	6·7	
7	95	76·0	6·75	6·75	
8	95	76·0	6·7	6·7	$\Delta = 60$ $C_1 = 2{\cdot}17$ $C_2 = 0{\cdot}96$
9	90	65·1	6·6	6·65	
10	85	54·3	6·2	6·4	
11	80	43·4	6·15	6·0	
12	75	32·6	5·0	5·35	
13	70	21·7	4·3	4·35	
14	65	10·9	3·1	2·9	

TABLE LIII.—*Experiment* 28.—TIGERSTEDT and WILLHARD.

No.	P	$C_1(P-\Delta)$	L. obs.	L. calc.	Const.
1	40	0·34	0·95	0·3	$\Delta = 39{\cdot}9$ $C_1 = 3{\cdot}44$ $C_2 = 0{\cdot}39$
2	42	7·2	2·6	3·4	
3	44	14·1	5·15	5·3	
4	46	20·5	6·65	6·65	
5	48	27·9	7·8	7·8	
6	50	34·7	8·9	8·65	
7	52	41·6	9·45	9·3	
8	54	48·5	9·8	9·8	
9	56	55·4	10·15	10·15	
10	58	62·3	10·4	10·35	
11	60	69·1	10·45	10·45	
12	62	76·0	10·5	10·5	
13	64	82·9	10·6	10·45	
14	66	—	10·65	—	
15	68	—	10·65	—	
16	70	—	10·7	—	
17	75	—	10·7	—	
18	80	—	10·7	—	
19	85	—	10·75	—	
20	85	—	10·8	—	$\Delta = 40{\cdot}6$ $C_1 = 3{\cdot}55$ $C_2 = 0{\cdot}36$
21	80	—	10·85	—	
22	75	—	10·85	—	
23	70	—	10·85	—	
24	68	—	10·9	—	
25	66	90·12	10·85	10·7	
26	64	83·1	10·9	10·87	
27	62	76·0	10·9	10·9	
28	60	68·6	10·9	10·9	
29	58	61·5	11·05	10·8	
30	56	54·4	10·75	10·5	
31	54	47·4	10·4	10·1	
32	52	40·3	10·0	9·55	
33	50	33·2	9·1	8·8	
34	48	26·2	8·0	7·9	
35	46	19·1	6·65	6·65	
36	44	12·0	4·9	4·95	
37	42	4·9	0·0	2·7	
38	40	0·0	0·0	0·0	

TABLE LIV.—*Experiment* 38.—TIGERSTEDT and WILLHARD.

No.	P	$C_1(P-\Delta)$	L. obs.	L. calc.	Const.
1	55	19·2	5·8	5·85	$\Delta = 48{\cdot}2$ $C_1 = 2{\cdot}84$ $C_2 = 0{\cdot}51$
2	60	33·4	7·8	7·8	
3	65	47·6	8·8	8·95	
4	70	61·8	9·4	9·5	
5	75	76·0	9·65	9·65	

TABLE LV.—*Experiment* 32A.—TIGERSTEDT and WILLHARD.

No.	P	$C_1(P-\Delta)$	L. obs.	L. calc.	Const.
1	4·1	5·7	0·5	3·65	$\Delta = 1{\cdot}5$ $C_1 = 2{\cdot}18$ $C_2 = 0{\cdot}48$
2	7·7	13·5	6·65	6·65	
3	10·9	20·5	8·85	8·55	
4	14·4	28·1	10·2	10·1	
5	18·1	36·1	11·0	11·3	
6	21·6	43·8	12·0	12·15	
7	25·2	51·6	12·2	12·8	
8	28·8	59·4	13·0	13·2	
9	32·8	68·1	13·4	13·4	
10	36·4	76·0	13·5	13·5	
11	36·4	76·0	14·4	13·8	$\Delta = 0{\cdot}5$ $C_1 = 2{\cdot}12$ $C_2 = 0{\cdot}50$
12	32·8	68·4	13·75	13·75	
13	28·8	59·9	12·9	13·55	
14	25·2	52·3	12·65	13·1	
15	21·6	44.7	12·4	12·5	
16	18·1	37·3	11·4	11·65	
17	14·4	29·4	10·8	10·5	
18	10·9	22·0	9·7	9·05	
19	7·7	15·2	7·1	7·3	
20	4·1	7·6	1·15	4·6	

TABLE LVI.—*Experiment* 2.—Von Kries.

No.	P	$C_1(P-\Delta)$	L. obs.	L. calc.	Const.
1	17	0·0	3·0	0·0	$\Delta = 17$ $C_2 = 0·25$ $C_1 = 5·07$
2	18	5·1	3·0	2·75	
3	20	15·2	5·5	5·8	
4	22	25·3	6·0	7·75	
5	24	35·5	9·0	9·1	
6	26	45·6	10·0	10·0	
7	28	55·7	10·2	10·5	
8	32	76·0	11·1	10·95	

No.	P	$C_1(P-\Delta)$	L. obs.	L. calc.	Const.
8	32	76·0	11·1	11·1	$\Delta = 17·25$ $C_2 = 0·24$ $C_1 = 5·15$
9	28	55·4	10·7	10·75	
10	26	45·1	10·1	10·1	
11	24	34·8	8·8	9·15	
12	22	24·5	7·0	7·7	
13	20	14·2	5·5	5·6	
14	18	3·9	2·6	2·3	
15	17	0·0	2·6	0·0	

TABLE LVII.—*Experiment* 3.—Cybulski and Zanietowski.

Isotonic Series.

No.	P coulombs	L. obs.	No.	P coulombs	L. obs.
10	$25·4 \times 10^{-10}$	29·30	19	33·4	32·18
11	27·4	29·64	20	33·5	32·54
12	29·6	29·98	21	33·6	32·81
13	30·8	30·30	22	33·7	33·07
14	32·6	30·60	23	33·8	33·43
15	33·0	30·96	24	33·9	33·74
16	33·1	31·29	25	34·0	34·01
17	33·2	31·60	26	34·1	34·32
18	33·3	31·92	27	34·2	34·41

TABLE LVIII.—*Experiment* 2.—Cybulski and Zanietowski.

Isotonic Series.

No.	P coulombs	$C_1(P-\Delta)$	L. obs.	L. calc.	Const.
1	$26·3 \times 10^{-11}$	0·0	1·0	0·0	$\Delta = 26·6$ $C_2 = 0·034$ $C_1 = 14·34$
2	26·80	2·86	3·8	4·65	
3	27·17	8·17	8·6	10·05	
4	27·53	13·34	14·8	13·95	
5	27·90	18·64	16·2	17·0	
6	28·30	24·38	19·6	19·8	
7	28·60	28·68	20·2	21·5	
8	28·96	33·84	22·4	23·15	
9	29·30	38·72	23·0	24·55	
10	29·60	43·02	26·2	25·55	
11	29·98	48·57	26·4	26·55	
12	30·30	53·16	26·5	27·2	
13	30·60	57·36	28·0	27·75	
14	30·96	62·62	28·2	28·15	
15	31·29	67·35	28·3	28·4	
16	31·60	71·70	28·4	28·45	
17	31·90	76·00	28·5	28·5	
18	32·18	80·3	28·6	28·4	
19	32·50	84·6	28·7	28·3	
20	32·80	—	28·8	—	
21	33·07	—	28·9	—	
22	33·43	—	29·0	—	
23	33·70	—	29·2	—	
24	34·01	—	29·2	—	

TABLE LIX.—*Experiment* 6.—CYBULSKI and ZANIETOWSKI.

ISOTONIC SERIES.

No.	P coulombs	C_1 (P − Δ) (A.)	L. obs.	L. calc.	Const.
1	10×10^{-10}	0·0	0·25	0·0	
2	10·95	0·0	4·0	0·0	
3	11·85	5·87	6·1	7·25	
4	12·65	11·40	11·6	11·5	
5	13·40	16·58	14·8	14·6	
6	14·15	21·76	16·7	17·0	
7	14·85	26·60	20·3	18·95	
8	15·50	31·09	21·5	20·4	
9	16·10	35·24	21·6	21·6	
10	16·75	39·73	22·6	22·65	Δ = 11·0
11	17·30	43·53	22·9	23·45	C_1 = 0·91 C_2 = 0·078
12	17·90	47·67	24·3	24·15	
13	18·45	51·47	25·0	24·7	
14	18·95	54·93	25·2	25·1	
15	19·50	58·73	25·4	25·4	
16	20·00	62·18	25·6	25·7	
17	20·50	65·64	25·8	25·9	
18	20·95	68·74	26·0	26·0	
		(B.)			
1	$14·15 \times 10^{-10}$	0·0	0·25	0·0	
2	14·85	0·0	0·25	0·0	
3	15·50	8·0	7·6	9·5	
4	16·10	12·8	12·8	13·0	
5	16·75	18·0	16·5	16·05	
6	17·30	22·4	17·7	18·15	
7	17·90	27·2	19·7	20·0	Δ = 14·8
8	18·45	31·6	21·0	21·6	C_1 = 8·0 C_2 = 0·064
9	18·95	35·6	22·4	22·8	
10	19·50	40·0	24·0	23·9	
11	20·00	44·0	24·9	24·75	
12	20·50	48·0	25·1	25·4	
13	20·95	51·6	26·0	26·0	

TABLE LX.—*Experiment of* CYBULSKI and ZANIETOWSKI.

ISOTONIC SERIES.

Curve I (*Twitch*).

No.	P coulombs	C_1 (P − Δ)	L. obs.	L. calc.	Const.
1	$37·41 \times 10^{-10}$	0·0	0·25	0·0	
3	38·73	0·0	0·75	0·0	
5	40·00	2·92	1·5	1·5	Δ = 39·8
7	41·23	20·90	4·0	5·75	C_1 = 14·62
9	42·43	38·44	7·5	7·7	C_2 = 0·106
11	44·72	71·90	9·0	9·0	

Curve II (*Tetani*).

2	$37·41 \times 10^{-10}$	0·0	0·75	0·0	
4	38·73	6·62	8·0	8·1	
6	40·00	14·40	13·5	13·6	Δ = 37·65
8	41·23	21·95	16·5	17·4	C_1 = 6·13
10	42·43	29·30	20·0	20·2	C_2 = 0·085
12	44·72	43·34	24·0	23·9	

TABLE LXI.—*Experiment of* WALLER.

No.	P	C_1(P − Δ)	L. obs.	L. calc.	Const.
1	1·52	0·0	0·0	0·0	
2	1·65	7·24	4·0	4·1	
3	1·79	24·13	9·0	8·7	Δ = 1·59
4	1·97	45·84	12·0	11·6	C_1 = 120·6
5	2·13	65·15	12·5	12·5	C_2 = 0·0085
6	2·32	88·07	12·5	12·4	

TABLE LXII.—*Experiment* 6.—KEITH LUCAS.

No.	P Defl. galv.	$C_1(P-\Delta)$	L. obs.	L. calc.	Const.
1	39·45	16·7	0·0	3·84	
2	39·85	20·6	3·36	4·34	
3	40·25	24·5	4·96	4·77	
4	40·65	28·4	5·12	5·14	
5	41·05	32·4	5·44	5·47	
6	41·45	36·3	5·46	5·74	
7	41·85	40·2	5·47	5·98	
8	42·25	41·1	6·28	6·18	
9	42·65	48·1	6·37	6·35	
10	43·05	52·0	6·39	6·49	
11	43·50	56·4	6·10	6·63	
12	44·00	61·3	6·72	6·73	
13	44·50	66·2	6·74	6·81	
14	45·00	71·1	6·76	6·83	
15	45·50	76·0	6·85	6·85	
16	46·00	80·9	6·83	6·82	
17	46·50	—	6·85	—	
18	47·00	—	6·86	—	
19	52·00	—	6·88	—	
20	58·00	—	6·89	—	
21	76·25	—	6·88	—	
22	100·25	—	6·87	—	
23	140·00	—	6·90	—	
24	200·00	—	6·88	—	

$\Delta = 37·75$ $C_2 = 0·208$ $C_1 = 9·81$

TABLE LXIII.—*Experiment* 11.—GILDEMEISTER.

CURVE I.

No.	P	$C_1(P-\Delta)$	L. obs.	L. calc.	Const.
1	15	14·9	8·75 9·25	10·0	
3	16	21·7	12·25 12·50	12·45	
5	17	28·5	15·25 15·00	14·35	
7	18	35·3	16·00 16·00	15·80	
9	19	42·1	16·75 17·00	16·95	
11	20	48·8	17·75 17·50	17·80	
13	25	82·8	19·00 19·25	19·00	
15	30	116·7	18·75 18·75	16·42	
17	40	—	19·00 19·00	—	
19	50	—	19·00 19·25	—	
21	60	—	19·00 19·00	—	
23	70	—	19·00 19·00	—	
25	80	—	19·00 19·25	—	
27	90	—	19·00 19·00	—	
29	100	—	19·00 19·00	—	

$\Delta = 12·8$ $C_2 = 0·108$ $C_1 = 6·78$

CURVE II.

No.	P	$C_1(P-\Delta)$	L. obs.	L. calc.	Const.
2	15	0·6	0·0	1·15	
4	16	1·1	0·0	2·02	
6	17	1·7	0·0	2·81	
8	18	2·2	0·0	3·52	
10	19	2·8	0·0	4·16	
12	20	3·3	0·0	4·79	
14	25	6·1	0·0	7·49	
16	30	8·9	0·0	11·36	
18	40	14·5	11·00 11·25	13·3	
20	50	20·1	16·00 16·25	16·15	
22	60	25·7	19·00 19·00	18·45	
24	70	31·3	20·5 20·5	20·30	
26	80	36·9	21·25 21·00	21·80	
28	90	42·5	22·75 23·00	23·00	
30	100	48·1	24·00 24·00	24·00	

$\Delta = 14$ $C_2 = 0·067$ $C_1 = 0·56$

TABLE LXIV.—*Experiment* 2.—MAY.

No.	P	C_1 (P − Δ)	L. obs.	L. calc.	Const.
1	20	7·86	17·5	14·2	Δ = 5 C_1 = 0·52 C_2 = 0·65
2	35	15·72	22·5	22·35	
3	50	23·58	27·0	28·15	
4	60	28·25	30·5	30·85	
5	70	34·07	33·0	33·7	
6	80	39·31	36·0	35·8	
7	90	44·55	37·0	37·5	
8	100	49·79	38·0	38·8	
9	110	55·03	38·5	39·65	
10	120	60·27	40·5	40·4	
11	130	65·51	40·5	41·0	
12	140	70·75	41·25	41·2	
13	150	76·00	41·3	41·3	
14	160	81·24	40·5	41·0	
15	170	86·48	40·0	40·6	
16	180	91·72	40·0	40·1	
17	190	96·96	39·5	39·5	
18	200	102·20	38·0	38·5	

TABLE LXV.—*Experiment* 3.—MAY.

No.	P	C_1(P − Δ)	L. obs.	L. calc.	Const.
1	20	13·79	9·5	8·1	Δ = 0·5 C_1 = 0·71 C_2 = 1·23
2	35	24·75	11·75	11·25	
3	50	35·35	13·75	13·35	
4	65	45·95	14·75	14·75	
5	80	56·56	15·5	15·5	
6	100	70·70	15·75	16·0	
7	125	88·37	(?)	15·75	
8	150	106·05	14·75	14·75	
9	175	123·72	13·75	13·10	
10	200	141·40	11·5	11·20	

TABLE LXVI.—*Experiment* 4.—MAY.

No.	P	C_1(P − Δ)	L. obs.	L. calc.	Const.
1	20	8·14	28·0	20·8	Δ = 2 C_1 = 0·45 C_2 = 0·52
2	35	14·93	31·0	30·9	
3	50	21·72	38·5	38·5	
4	60	26·24	43·0	42·55	
5	70	30·76	46·25	46·0	
6	80	35·29	49·25	48·9	
7	90	39·81	51·50	51·4	
8	100	44·34	53·0	53·35	
9	110	48·86	54·5	55·1	
10	120	53·38	55·25	56·3	
11	130	57·91	56·25	57·4	
12	140	62·43	57·25	58·2	
13	150	66·96	58·50	58·75	
14	160	71·48	58·75	58·8	
15	170	76·00	59·0	59·0	
16	180	80·53	58·5	58·75	
17	190	85·05	58·0	58·15	
18	200	89·58	57·75	57·85	

UNMYELINATED NERVE-FIBRES AS CONDUCTORS OF PROTOPATHIC SENSATION.

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THE object of this paper is to bring together a number of scattered observations on the structure and function of the afferent fibres of the cerebrospinal nerves, and to formulate on the basis of these observations a working hypothesis, which should greatly facilitate further investigation along these lines. As yet we do not know how the sensations of touch, pain, and temperature are differentiated and how the underlying afferent impulses are propagated along the peripheral nerves.

PROTOPATHIC NERVE-FIBRES.

An important advance was made by Head and his associates (1905, 1906, 1908) when they showed that cutaneous sensations could be separated into two groups, to which they applied the terms "protopathic" and "epicritic." Under the term "protopathic," Head groups, in addition to pain, the temperature sensations aroused by objects under 22° C. or over 40° C. This group is characterized by a "peculiar tingling quality," by radiation into other parts than those stimulated, and by failure of the subject to localize accurately the point stimulated. Under the term "epicritic" he groups sensibility to light touch, temperature sensations derived from objects between about 22° C. and 40° C.,[1] and discrimination of the two compass points. Sensations of this kind are all accurately localized. It would take too much space to tell in detail how these two types of sensation were separated from each other. Obviously such a distinction could not be made by a study of the normal skin. It was found, however, that after lesions of the dorsal roots areas of pure epicritic sensation appeared. Such a cutaneous area was sensitive to light touch and to medium degrees of temperature, but insensitive to pain

[1] These are the approximate limits as determined in clinical work on divided peripheral nerves. In the experiment recorded by Rivers and Head these limits could be more accurately determined and were found to lie between 25° or 26° C., and 37° to 38° C.—[ED.]

and to the more extreme degrees of temperature. On the other hand, when the median nerve was cut a cutaneous field became outlined on the palm of the hand, in which only protopathic sensations were experienced. Here the skin was sensitive to pain and to the extreme degrees of temperature, but insensitive to light touch and the intermediate degrees of temperature. All sensations from such an area were poorly localized and had a peculiar tingling quality. Head and his associates studied a very large series of cases with nerve lesions and found many such areas of dissociated sensation.

I have myself had an opportunity of studying in one of my students a partially anæsthetic area in the palm of the hand, resulting from the division of the median nerve, and have found it to possess all the characteristics of a protopathic area. There is complete loss of sensibility to light touch over the entire area of distribution of the median nerve. But over a considerable portion of this area the skin is acutely sensitive to the prick of a pin. A test-tube filled with ice water was felt as cold and one filled with water at 50° C. as hot, but test-tubes with water at 22° C. and 40° C. were not distinguishable from each other. Sensations from this area were not well localized, and had a peculiarly unpleasant character. In other words, the area possessed sensation of a typically protopathic character.

It is maintained by Head that each of his two sensory groups depends on a separate, anatomically distinct, set of nerve-fibres. He presents good and convincing reasons for this belief, but space does not permit us to repeat the argument here. We wish only to call attention to a fact which might easily be overlooked in reading the original articles. In areas of partial anæsthesia the residual sensation may be either protopathic or epicritic. If the only form of residual sensation were protopathic, one might assume that it depended only on a decreased density of innervation. It might easily be that light touch required a denser innervation for its perception than pain. But the reverse form of partial anæsthesia also occurs (and numerous examples of it are given by Head) in which epicritic sensation persists over an area devoid of protopathic sensation. It is not conceivable that a simple decreased density of innervation should in the one case give rise to a loss of light touch with pain persisting, and in another case cause a complete loss of pain sense while light touch remains normal. Furthermore, these areas of pure epicritic sensation are sensitive to temperature between about 22° C. and 40° C., but insensitive to the more extreme degrees of temperature. This is clearly not a case of lowered sensibility

due to decreased density of innervation. It seems clear to me that these facts can be explained only on Head's assumption that there are two kinds of afferent nerve-fibres, which differ slightly in their anatomical distribution.

According to Head the unit of distribution of protopathic fibres is the dorsal root, each root having a sharply outlined area of skin which it supplies with them. The epicritic fibres of adjacent roots are intermingled in their cutaneous distribution. Section of one or more dorsal roots deprives a sharply circumscribed area of skin of its protopathic fibres, while epicritic fibres from adjacent roots run into this area, endowing more or less of the skin near its border with pure epicritic sensation. Here light touch is felt, but not pain, warm and cold objects are discriminated, but hot and cold objects give rise to no temperature sensation. In the same way the peripheral nerve is the unit of epicritic sensation. The epicritic fibres of the ulnar nerve are limited to the area of skin outlined by anatomists as representing the cutaneous distribution of that nerve; but the protopathic fibres of the ulnar run long distances in the subcutaneous plexuses into the areas belonging to adjacent nerves. When the median nerve is cut epicritic sensation is lost over the entire area ordinarily assigned by anatomists to that nerve, but protopathic fibres from the ulnar nerve run into this area endowing a considerable extent of the skin near the border of the area with pure protopathic sensation. Here pain is felt but not touch. Hot and cold objects are distinguished, but warm and cool objects give rise to no temperature sensation.

Facts which are otherwise inexplicable are thus readily understood on the assumption of two kinds of nerve-fibres which vary slightly in their anatomical distribution. This assumption acquires still greater significance in view of the recent demonstration that there are two kinds of afferent cerebrospinal nerve-fibres which differ both in structure and distribution. Until recently we were acquainted with only one of these, the myelinated fibres. But Ranson has recently shown that there are great numbers of unmyelinated afferent fibres that had previously been overlooked. Can it be that one kind, say the myelinated, is responsible for epicritic sensation and the other, the unmyelinated, for protopathic sensation? In the résumé which follows we shall see that there is the most striking parallelism between what is known of the protopathic fibres and what has recently been determined in regard to the unmyelinated afferent nerve-fibres.

Unmyelinated Afferent Nerve-fibres.

Ranson (1912) modified the Cajal silver technique in such a way as to produce a differential stain of unmyelinated fibres, and applied to the method the term "pyridine silver technique." When the spinal nerves are stained by this method they are seen to contain great numbers of unmyelinated fibres. These are, in fact, more numerous than those which are myelinated. It is possible to trace them centrally and to show that they arise from the small cells of the spinal ganglia. These small spinal ganglion cells possess single axons which divide dichotomously into fibres running into the peripheral nerves on the one hand and into the dorsal roots on the other. These axons and their branches remain unmyelinated throughout their course. Fibres of this kind are also present in the cranial nerves, particularly in the vagus, where they can be demonstrated with the greatest ease (Chase and Ranson, 1914, Ranson, 1914). The two kinds of nerve-fibres seem to be sharply separately from each other with few or no transition forms in the shape of partially myelinated fibres. That the distinction between the two is a fundamental one is evidenced by the fact that they are present in the same relative proportion and distributed in the same way in the vagus nerve of a reptile (the snapping turtle) as in that of a mammal (the dog) (Ranson, 1915). It would be strange if such a sharp division of afferent nerve-fibres into two kinds according to structure did not run parallel to the equally sharp division of these fibres according to function. We should, therefore, expect to be able to assign protopathic sensation to one and epicritic to the other. In fact, we shall see that the protopathic and unmyelinated fibres have a great many things in common.

Distribution of the afferent unmyelinated fibres.—The unmyelinated fibres of spinal nerves are distributed chiefly to the skin, but some go also with the muscular branches of the nerves. On the assumption that fibres of this kind mediate protopathic sensation those going to the deeper structure would account for the sense of pain in the muscles, joints and other deeply situated parts. The assumption that the fibres carrying cutaneous pain belong to a different class from those carrying pain from the muscles and joints is a weakness in the theory as presented by Head which we are now able to avoid.

The unmyelinated fibres are not distributed equally in all cutaneous nerves. The median nerve at the wrist contains an absolutely and relatively much greater number of myelinated nerve-fibres than does

the medial cutaneous nerve of the forearm. The former is distributed to the skin of the fingers which possesses a high order of epicritic sensation, and it is composed predominantly of myelinated fibres; the latter is distributed to an area whose epicritic sensation is of a lower order, and it contains correspondingly fewer myelinated fibres so that the unmyelinated fibres greatly predominate. The lateral femoral cutaneous nerve is another instance of a nerve running to an area of low epicritic sensibility and containing relatively few myelinated fibres. There seems, therefore, to be a direct relation between the sharpness of epicritic sensation and the proportion of myelinated fibres in the associated nerve.

Rate of regeneration.—As yet we have no information regarding the relative rate of regeneration of the myelinated and unmyelinated afferent nerve-fibres. Head has shown, however, that after division and suture of a nerve protopathic sensation returns to the area supplied by that nerve much more quickly than does epicritic sensation. Protopathic sensation may be completely restored over the entire area supplied by the regenerating nerve for months before epicritic sensation begins to return. Now it is a significant fact that exactly at the time at which protopathic sensation is restored innervation returns to the sweat glands and blood-vessels. This means a regeneration of the vasomotor and secretory fibres, which are post-ganglionic autonomic (or sympathetic) fibres. These are known to be unmyelinated. On the assumption that the protopathic fibres are also unmyelinated the fact that these two groups of fibres complete their regeneration at the same time would be easily understood.

Visceral sensation.—In addition to sensations of movement and pressure the viscera give rise to sensations of a protopathic character. Sensations of movement and pressure are attributed by Head to the large myelinated visceral afferent fibres which run through the sympathetic system to end in pacinian corpuscles. The stomach and its mucosa are insensitive to touch. While under ordinary conditions sensations from the stomach are not recognized in consciousness, yet it is a matter of common experience that under certain conditions pain may be felt in that viscus. Carlson (1914) has shown that the mucosa of the stomach is sensitive to temperatures of 10° and 50° C., but insensitive to the intermediate degrees. He concludes that it is endowed with protopathic temperature sensation. In the same way Head showed that the colon was sensitive to temperatures in the protopathic range. The evidence, therefore, supports Head's contention that the viscera possess protopathic but not epicritic sensibility.

Now it has been shown that the afferent fibres of the vagus coming from the stomach are chiefly unmyelinated. The vagus nerve, as it pierces the diaphragm, is practically an unmyelinated nerve (Chase and Ranson, 1914). It contains relatively fewer myelinated fibres than many sympathetic nerves, but is solidly packed with unmyelinated axons. Molhant (1913) has shown that about one-quarter of the cells of the nodose ganglion give rise to fibres running to the stomach. This is an enormous number of cells, only a small part of which could be accounted for by the few myelinated fibres in the gastric rami, even if one overlooked the fact that at least some of these are probably visceral efferent fibres. It is therefore clear that the main sensory innervation of the stomach is with unmyelinated fibres, and that, here, a very close parallel exists between the distribution of these fibres and those mediating protopathic sensation.

Course of the afferent fibres in the spinal cord.—It is well known that the afferent impulses underlying sensations of pain and temperature pass through the grey matter and cross to the opposite side of the cord at or near the level at which they reach it. Head (1906) has shown that this is true for temperature sensation of the epicritic as well as of the protopathic order. According to him the other elements of the epicritic group (touch, tactile discrimination, and tactile localization) are carried upward on the same side of the cord in the posterior funiculus for varying distances before ending in the grey matter. He maintains that the tactile impulses coming in along a given root do not cross to the opposite side of the cord all at once, but that they ascend in the posterior columns for varying distances. The crossing at varying levels of impulses coming in by a single root gives rise to a double pathway for touch, uncrossed fibres of the first order paralleling crossed fibres of the second order for a certain number of segments. This double path no doubt accounts for the conflicting observations on the conduction of tactile impulses which are found in the literature.

The facts that have been ascertained regarding the intraspinal course of the unmyelinated fibres is in complete accord with the view that they are the conductors of protopathic afferent impulses. As a dorsal root enters the spinal cord the two kinds of fibres separate, the unmyelinated turn laterally into Lissauer's tract while the myelinated run on into the posterior funiculus (Ranson, 1913, 1914). Few, if any, unmyelinated fibres enter that funiculus, but a few fine myelinated fibres run into the tract of Lissauer. This consists chiefly of unmyelinated axons scattered among which are a few fine myelinated

fibres. From the level at which they enter the cord, the fibres ascend or descend in this tract for a very short distance not exceeding one or two segments. The substantia gelatinosa seems to be the sensory nucleus associated with this tract.

The unmyelinated fibres, then, enter the grey matter at or near the level at which they enter the cord. In this they are again in exact agreement with the fibres conveying protopathic sensation. The myelinated fibres, which alone enter the posterior funiculus, correspond in their intramedullary course to the fibres carrying light touch, tactile discrimination and tactile localization, since, according to Head, these ascend for longer or shorter distances in this funiculus before entering the grey matter. As to the temperature sensation in the epicritic range, they are probably conveyed by the fine myelinated dorsal root-fibres that run with the unmyelinated ones into the tract of Lissauer. It is thus apparent that we have at hand data sufficient to explain the intramedullary course of the protopathic and epicritic sensation in terms of the demonstrated intramedullary course of the myelinated and unmyelinated fibres.

So far as the evidence goes the theory that protopathic sensation is conveyed by unmyelinated fibres seems to be well supported. A number of methods of testing this hypothesis have occurred to us and experiments directed to this end are now under way. Some of these have progressed far enough to give evidence of value.

The function of the tract of Lissauer.—Experiments on the spinal cord of the cat (Ranson and von Hess, 1915) have shown that the tract of Lissauer and the substantia gelatinosa Rolandi are at least closely associated with the pain reception and conduction apparatus. It was found that while bilateral destruction of the tract of Lissauer and the substantia gelatinosa at the level of the first lumbar segment of the cat's cord did not interfere in any way with the perception of pain in the hind limbs, it entirely eliminated the pressor vasomotor reflex from stimulation of the sciatic nerve. Now the vasomotor reflexes are distinctly protopathic in that they are produced almost exclusively by pain and temperature sensation. The evidence, presented in that paper, showed that the tract of Lissauer and the substantia gelatinosa formed a path for conduction of the afferent impulses involved in the reflex vaso-constriction due to painful sciatic stimulation. It seemed probable to us that the tract of Lissauer and the substantia gelatinosa Rolandi form an apparatus for the reception and intersegmental conduction of painful afferent impulses. Some impulses from this apparatus passing

over to the spinothalamic tract would reach the cortex and find expression as conscious pain, while other impulses received in this apparatus would ascend and descend within it, producing pain reflexes. So far as the evidence goes this work favours the theory that the unmyelinated fibres conduct protopathic sensation in that it shows that the portion of the cord in which these fibres run and terminate forms part of a protopathic reflex arc.

We hope to obtain more direct evidence from two lines of investigation which we are about to undertake. The glans penis is a region possessing only deep and protopathic sensibility, and if the conception presented in this paper is correct the nerve-fibres distributed to the skin of this region should be for the most part unmyelinated. Again, by experimental work on animals, it should be possible to find hyperæsthetic areas at the borders of anæsthetic areas resulting from peripheral nerve lesions. The nerve-fibres in such hyperæsthetic areas should, according to the view here presented, be for the most part of the unmyelinated variety. We propose to investigate both of these questions.

It is interesting to note that throughout his four papers dealing with this question Head conveys the impression that he regards the protopathic group of sensations as primitive in character and the first to appear in phylogenetic development. Now it is well known that nerve-fibres in their earliest phylogenesis are unmyelinated. If our conception is correct a very large part of the afferent nerve-fibres of mammals remain in this primitive relatively undifferentiated state and mediate a relatively primitive and diffuse form of sensation. Other afferent fibres have undergone further differentiation, acquired a myelin sheath, and carry more highly specialized forms of sharply localized sensation. Insulation of the axon by the myelin sheath may play a part in eliminating the element of diffusion so characteristic of the more primitive sensations.

In conclusion, we wish to state that we do not consider that we have done more than formulate a working hypothesis, and shall be satisfied if the considerations here presented stimulate further discussion and investigation.

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